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The President's New Year Message

It is customary in all countries to look forward with hope at the turn of the year, and I think particularly so where effort is required to overcome the vagaries of nature. The British Stock has met the challenge wherever and whenever it was to be found. The Veterinary Profession, epitomising this spirit, has a good record in its fields of endeavour, and its opportunities for furthering this record are ever increasing.

At this time of stocktaking and preparation for a new year it might be wise to check our equipment. If it is in good shape our hopes can be translated into fact, and if not, then our hopes remain as dreams. Our basic equipment is knowledge—knowledge of disease in all its aspects, opportunities for furthering this knowledge and disseminating it, and the machinery for integrating with other sections of the community.

I am satisfied that the fundamental knowledge and experience of the British Veterinary Profession is sound, and the opportunities for furthering it under the guidance of the R.C.V.S., the new University Schools, and the many Research Centres, will bring a steady stream of new facts. Future generations should be well equipped to meet their problems.

The integration of this knowledge for the benefit of all is largely the task of the B.V.A. through its Technical Development, Organising, and Parliamentary and Public Relations Committees, who have the machinery geared and running smoothly.

Relations with outside bodies and Government Departments have been built up on a basis of mutual respect, and I think it is true that the members of the Association have confidence in the Council. When faced with the need to invest capital in new headquarters, there was, therefore, complete agreement with the Premises Sub-committee's choice of No. 7, Mansfield Street. In this fine old Adam house the B.V.A. have offices of which they may well be very proud. A far cry from the 1920s when the N.V.M.A. first came into being. Then the far sighted, if optimistic hope of a few that this small profession would succeed in making a united advance with the constituent Divisions and a virile Scottish Branch in support. The Welsh Branch now in being, is also tangible evidence of a healthy Association.

We also have closer links with the Overseas Associations to our mutual benefit.

The Association is served by a loyal and experienced staff.

We can for the coming year, therefore, base our hopes on faith—faith in the knowledge of our ability to lead in the provinces of Agriculture or Science which by training and experience we are best fitted to do.

To members and friends I wish each and all a Happy and Prosperous New Year.

Alex Thomson

PROTRUSION OF THE INTERVERTEBRAL DISC IN THE DOG

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PART I.—INTRODUCTION

Locomotory paralysis in the dog has long been familiar to veterinarians in this country. Many features of it, its symptoms, course and prognosis, for example, are comparatively well known. Knowledge of its cause, on the other hand, is more limited. Generally, certain broad possibilities would be considered, notably trauma, distemper and other infectious nervous diseases, toxaemia, nutritional disturbance, etc. Occasionally other more specific possibilities have been suggested, such as calcification of the intervertebral disc. However, recent research abroad since 1946, mainly in Sweden and the United States, has produced new information and, especially, some new ideas on the aetiology. Throughout the latter the keynote has been protrusion of the intervertebral disc.

An initial interest in the functional anatomy of the vertebral column led the present authors to read the principal articles from these sources. At the same time it was found that the new concept of disc protrusion as a major cause of canine paraplegia had not been fully accepted in this country. In discussing, in 1949, the differential diagnosis of canine paraplegia, Boddie (1949) remarked that he had found no radiographic evidence of disc prolapse in the dog and that it was anatomically impossible for it to occur in a manner comparable to that seen in man. Kirk (1953) comments that it is still a subject of dispute.

Because of the lack of agreement it was decided to attempt to test for ourselves the main clinical and morbid anatomical observations described abroad. *Post-mortem* examinations, therefore, were made on a trial series of seven dogs showing locomotory paralysis but free from distemper and other comparable diseases and trauma. The result was that in each case there were findings typical of those described abroad, and no difficulty was found in attributing the paralysis to disc protrusion.

In the British literature, to the best of our knowledge, there is only one survey of disc protrusion as a factor in canine paraplegia ; this is in the latest edition of the " Index of Diagnosis " by Kirk (1953). Chiefly in the section on changes in intervertebral discs this gives a clear description of many of the main features of disc protrusion. However, the principal objective of Kirk's text is, of course, diagnosis, and a number of aspects not particularly relevant to diagnosis are not included. Examples are the histopathology of the spinal cord, the results of surgical treatment, the anatomy of the conjugal ligament and its influence on the distribution of protrusions, and the full background of age and breed factors in the different types of disc degenerations. Yet each one of these aspects contributes valuable evidence in support of the new concept.

Because of the lack of a comprehensive account in the British literature and the continuation of controversy the need was felt for a review of the subject. It was thought that such a review should be sufficiently full to cover as many as possible of the aspects which might be relevant to the controversy ; this has been attempted in the following article. It will be evident that with this approach a full review of all the literature has not been undertaken ; however, we have tried to do justice to all articles in the English language dealing specifically with disc protrusion in the dog.

The remaining contents of the article are as follows : Part II, Review of the literature ; Part III, Two representative case reports ; Part IV, Discussion ; Part V, Summary.

PART II.—REVIEW OF LITERATURE

History

Since Dexler's work (1896), in which he described symptoms of posterior paralysis in the dog caused by compression of the cord which could be related to a chondrification of the intervertebral disc, this disease has been called enchondrosis intervertebralis. The condition is described under this name in many modern texts, *e.g.*, by Hutyra, Marek & Manninger (1949). Hansen (1951) showed this chondrification to be part of the process of disc degeneration and so enchondrosis intervertebralis becomes absorbed in this new concept. In the half-century between the publications of Dexler and Hansen several workers have attempted explanations of the pathology and pathogenesis of this disease which causes symptoms varying from pain to paraplegia. It is not until 1939 that we find Tillmans (1939) in Germany suggesting that protrusions found in the vertebral canal in these cases might be prolapsed disc tissue. The earliest mention that we can find in the English language of disc prolapse* in the dog appears in the Proceedings of the Royal Society of Medicine, 1939. In presenting a paper on protruded intervertebral discs in the human, Love (1939) gave a few details which had been supplied by Schlotthauer of a case occurring in the dog. In 1940 this case was quoted more fully by Schlotthauer in an account of the pathology of certain lesions of the nervous system in which he stated : " A prolapsed intervertebral disk was observed by me in a dog. It caused paralysis of the hind legs and urinary and anal sphincters. The symptoms were progressive in development." In 1946 Riser presented a paper to the Indiana Veterinary Medical Association and stated his belief that " protrusion of one or more of the intervertebral discs into the spinal foramen is a major contributing factor in many of these cases " (of posterior paralysis). It now seems to be accepted in America that intervertebral disc protrusion is a clinical entity (*vide* " Canine Surgery " (Lacroix & Hoskins, 1949)). Nevertheless, in 1950 Schnelle (1950) appeared to infer that calcification follows the " slipping " of the whole disc and also that the conformation of the dog is one of the responsible factors. On the Continent Fankhauser (1948) believed that the disc neoformations might consist of protruded nucleus pulposus tissue. In this country it is not until 1953 that we find Kirk in " Index of Diagnosis " describing prolapse as a change in the intervertebral disc and outlining the resulting symptoms. However, it appears that he considers disc calcification to be a cause of paralysis and possibly also a criterion of disc prolapse. Reports have appeared elsewhere in the British Press linking disc calcification with paralysis (Holmes, 1942 ; Kirk, 1942).

Associated with Hansen's work is that of Olsson and either together or separately from 1951 onward they have contributed several articles on the condition of disc degeneration and allied phenomena (Hansen, 1951, 1952 ; Hansen & Olsson, 1953 ; Olsson, 1951a, b ; Olsson & Hansen, 1952). Recently Hoerlein (1952, 1953) has produced a thesis and articles in the American veterinary Press embodying his work on the clinical and pathological aspects of disc protrusions in the dog.

* In this article prolapse and protrusion are considered to be synonymous.

Normal Anatomy of the Intervertebral Disc

The following account agrees with the standard texts on the anatomy of the dog. There are 27 vertebrae between the head and the sacrum. With the exception of the first two, these vertebrae are separated from each other by intervertebral discs which are very closely connected to the vertebral bodies. Immediately dorsal to the vertebral bodies is the vertebral canal which houses the spinal cord and its investing membranes. (For this and other anatomical features described below see Fig. 1.) Between the outermost of these membranes (dura mater) and the vertebral bodies is the dorsal longitudinal ligament which starts at the odontoid process of the second cervical vertebra and ends at the body of the first coccygeal vertebra. As it passes over each intervertebral disc it expands laterally and blends with the disc tissue (see Plate I). In the thoracic region a series of ligaments is interposed between the discs and dorsal longitudinal ligament, the conjugal ligaments. These connect the articular heads of corresponding ribs from the 2nd to the 10th. (Hansen (1952) says 2nd to 9th ribs ; there may well be a breed difference.) The ventral longitudinal ligament runs along the ventral aspect of the vertebral bodies but does not extend as far cranially as the dorsal ligament.

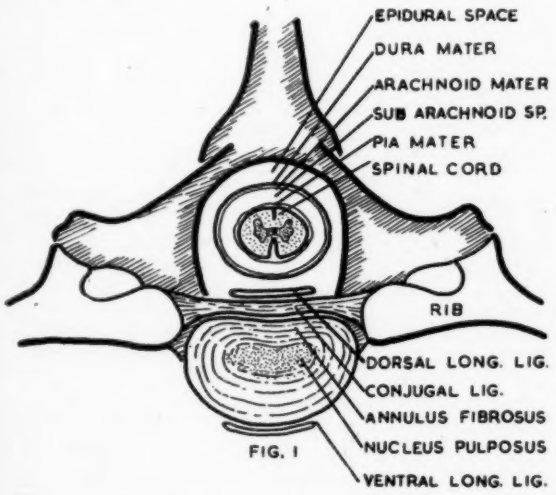

Fig. 1.—Diagrammatic Transverse Section through a Normal Mid-thoracic Intervertebral Disc

This shows the general anatomy of the neural canal and its contents and the disposition of the nucleus pulposus, annulus fibrosus, conjugal ligament, and dorsal longitudinal ligament (the last being in T.S.). The conjugal ligament is only present from the 2nd to the 9th or 10th rib. The vertebra is diagonally hatched.

Each disc consists of an outer fibrous ring, the annulus fibrosus, and a central jelly-like mucoid mass, the nucleus pulposus. The annulus fibrosus is usually much broader ventrally than dorsally and is markedly lamellated. Some of the fibres in its construction are direct continuations of the Sharpey's fibres of the vertebral bodies. The nucleus pulposus is a remnant of notochordal tissue which has now become highly specialised to act as a " shock-absorbing water cushion."

Most of the epidural space is filled with fat but venous plexuses are also present. Following the enlargement at the origin of the lumbo-sacral plexus the spinal cord becomes smaller in cross-section as it runs caudally ; the amount of fat in the epidural space increases and the distance between the floor of the vertebral canal and the cord becomes greater.

Pathology of the Intervertebral Disc

Degenerative changes may occur in the annulus fibrosus and the nucleus pulposus. Rupture of the degenerated fibres and protrusion of the nucleus pulposus tissue is then facilitated. It is difficult to decide when a disc is pathologically degenerate as some changes are due to normal ageing. Prolapse of disc material presupposes degeneration and, as Hansen (1952) points out, prolapses and calcifications are the only macroscopic expressions of degeneration unless the other types of degeneration cause an interruption in the continuity of the tissues. These macroscopic changes can now be examined in more detail.

Rupture of the Intervertebral Disc.—This signifies a partial or complete breakdown of the fibres of the annulus fibrosus. Hansen (1952) finds that partial ruptures are more common in the dorsal part of the annulus fibrosus than in the ventral. This author (1952) also contributes a case where protrusion of the nucleus pulposus had occurred into the body of the vertebra immediately caudal.

Ventral Displacement of the Nucleus Pulposus.—According to Hansen (1952) degenerative changes of the ventral part of the annulus fibrosus are usually found in the condition of Spondylitis (Spondylosis) Deformans. As is well known, this disease is a form of spinal ankylosis in which osteophytes are found either locally or generally, usually on the ventral surface in the region of the intervertebral discs of the thoracic and lumbar parts of the vertebral column. Hansen (1952) assumes that a ventral rupture of the annulus fibrosus with prolapse of disc material might be the probable precursor of spondylitis deformans, and illustrates one such case. Debard (1950) also attributes these osteophytes to displacement of the nucleus pulposus ventrally. This damages the attachment of the fibres of the annulus fibrosus to the vertebral bodies and may also damage the periosteum and ventral longitudinal ligament. The reaction to this damage leads to osteophyte production.

Dorsal Displacement of the Nucleus Pulposus.—It is apparent from the different descriptions of disc pathology that dorsal ruptures of the annulus fibrosus may vary in extent from slight degeneration and breakdown of fibres, allowing a small bulge upward of the still intact dorsal surface of the disc (Fig. 2), to complete rupture of the annulus fibrosus and ejection of nucleus pulposus. The route of the prolapse may be direct and easily visible on transverse section of the disc, or it may be thin and tortuous, traceable only by serial sections (Hansen, 1952). In some cases lifting of the dorsal longitudinal ligament will occur (Fig. 3) ; in other cases the protrusion may occur beside the longitudinal ligament (Fig. 4) ; and in yet other cases the material may split the dorsal longitudinal ligament to impinge on the dura mater (Fig. 5). If in any of these conditions the spinal cord should be damaged, either directly (as by contact with the protrusion), or indirectly (for instance by haemorrhages in the spinal canal), then it is to be expected that nervous symptoms will probably result. However, protrusions can occur without nervous sequelae. Any one or more of the intervertebral discs may rupture, but dorsal ejection of nucleus pulposus tissue is rarely reported from those discs covered by a conjugal ligament. It would seem that this ligament acts as a reinforcing band to the dorsal annulus (Hansen, 1952 ; Hoerlein, 1953).

No sex disposition seems to exist, but the frequency of disc prolapses grows with the age of the animals (Hansen,

FIG. 2

FIG. 3

FIG. 4

FIG. 5

FIGS. 2 TO 5.—DIAGRAMS OF VARIETIES OF DORSAL PROTRUSIONS

The discs are cut in transverse section, and are typical of the cervical, first and last three or four thoracic, and lumbar vertebrae. In the other thoracic vertebrae the conjugal ligament separates the disc from the dorsal longitudinal ligament as shown in Fig. 1. The arrow shows the route of the protrusion.

FIG. 2.—Partial rupture of annulus fibrosus.

FIG. 3.—Complete rupture of annulus fibrosus, but disc material confined beneath intact dorsal longitudinal ligament.

FIG. 4.—Complete rupture of annulus fibrosus lateral to edge of dorsal longitudinal ligament, the disc material escaping into neural canal.

FIG. 5.—Complete median rupture of annulus fibrosus and splitting of the dorsal longitudinal ligament, the disc material escaping into the neural canal.

1952 ; Hoerlein, 1953). The earliest clinical case Hansen (1952) records is at three years of age ; on an age-incidence graph Hoerlein (1953) shows three occurring at one year. Although dogs of nearly all breeds are reported as suffering from disc protrusion, Hansen (1952) points out that there is a definite breed incidence, the dachshund, French bulldog and Pekinese being most commonly affected. The cocker spaniel and beagle are also frequently subjected to the condition (Hoerlein, 1953) and McGrath (1951) considers " terriers " to be commonly affected. The sealyham and bulldog are also susceptible according to Kirk (1953).

The breeds mentioned by Hansen (1952) are generally recognised as chondrodystrophic (achondroplasic). This worker finds that in non-chondrodystrophoid breeds the nucleus pulposus matures by gradual increase of collagen fibres so that the mucoid material gradually becomes fibroid. Only in dogs above the age of seven is one more likely to find a fibroid than a mucoid nucleus pulposus. Although some macroscopic signs of disc degeneration have been found in individuals less than seven years of age, degeneration of the disc usually occurs in quite old age and then takes on the form of a necrosis. Calcified discs are very seldom seen in dogs of non-chondrodystrophoid breeds.

Hansen (1952) has also shown that in the chondrodystrophoid group it is usual for chondroid material to replace the mucoid nucleus pulposus by the age of one year. A partial or total dystrophic calcification of the nucleus pulposus is the most usual type of disc degeneration which follows in these breeds and in spaniels. This change is localised to the centre of the disc (nucleus pulposus and inner layer of the annulus fibrosus) and has been observed in puppies as young as five months of age. In dogs of this group above the age of two the probability of finding calcified discs is greater than 50 per cent.

CALCIFICATION OF THE INTERVERTEBRAL DISC.—Olsson (1951a) claims calcification of the disc to be a frequent phenomenon in protruded discs, but points out that calcification is also common in those discs that do not show the slightest bulge toward the vertebral canal. He agrees with Pommer (whom he quotes) that *no importance should be attached to calcification other than that it is a sign of degeneration.* Nevertheless, as Hoerlein points out, it is as well to remember that since a calcified disc is degenerate it is more liable to rupture and allow protrusion of disc tissue.

SYMPTOMS OF DORSAL DISC PROTRUSION

Olsson (1951a) separates his series of cases into several symptomatic categories. Hoerlein (1953) divides the symptom-complexes into fewer (four) major groups and so we have incorporated Olsson's findings into these groups where applicable.

1. *Pain.*—This may be abdominal, lumbar, or generalised. The animal may object to moving and will sometimes awaken frequently and cry out at night. Reflexes may be exaggerated and the muscle movements are usually spastic. Some degree of kyphosis or scoliosis is also sometimes seen. These symptoms may last for one or several weeks and there may be long intervals without symptoms. Olsson & Hansen (1952) point out that pain seems predominant with cervical disc lesions and that the animals often show a " psychic depression." Olsson (1951a) claims that those pain symptoms which give rise to a diagnosis of rheumatism and myalgia should be considered as due to disc protrusions.

2. *Paresis and/or Inco-ordination.*—Pain may be present in this state, and also a general hyperaesthesia. If the lesion is to one side, a unilateral paresis or a dragging of the toe or extended limb may result. Cervical lesions may give rise to paresis of all limbs. Paresis may follow a history of the pain complex of about a week or even several weeks.

3. *Paraplegia, Spastic or Flaccid.*—In a spastic paraplegia there are exaggerated reflexes and sensation and the animal carries his hind legs under him. Pain is usually present and also retention of urine and faeces. If the paraplegia is flaccid the muscles lack tone and the reflexes and sensations are diminished. There is incontinence of urine and faeces and the legs are dragged behind the body. The history may contain descriptions of one of the previous syndromes existing for a comparatively long time (days) or for a very short while (hours).

4. *Progressive Ascending Paralysis from Acute Disc Prolapse.*—A sudden onset characterises this complex and may be associated with great physical strain. Complete sensory and motor paralysis of the hind part of the body, together with urinary and faecal incontinence, is rapidly followed by an ascending paralysis resulting in a diaphragmatic respiration. The respiratory rate may be accelerated to such a point that the animal pants continuously (Riser, 1946). Death usually results from respiratory failure due to diaphragmatic or bulbar paralysis.

Although these symptom complexes often appear as separate entities, in chronic cases of disc protrusion the order of symptoms is usually pain, inco-ordination, spastic paraplegia, and finally flaccid paralysis. Apart from these drastic defects the dog may be quite normal (Hoerlein, 1953).

Acute cases are common in the chondrodystrophoid breeds

and may be connected with some form of strain (*e.g.*, manipulation). Although paralysis may be immediate the onset may be slow and a decrease in the symptoms followed by some degree of recovery is not unusual. Relapses are frequent, the interval between attacks varying greatly in extent (Hansen, 1952).

Diagnosis

According to Riser (1946) " the clinician who is acquainted with the symptoms has little trouble regarding the disease." Although the typical history and symptomatology do suggest a general diagnosis of disc protrusion, to obtain more specific information as to the site and extent of damage one must resort to radiography (Hoerlein, 1953). Normal (as opposed to contrast) radiography can be used to diagnose a good proportion of lesions (Olsson (1951a) mentions about half, Hoerlein (1953) about one-third).

Radiographs are taken laterally and ventro-dorsally (dog with its back on the X-ray plate). Hoerlein (1953) suggests a dorso-ventral position, but from principles of radiography one would expect the reverse position to give a more accurate picture.

From normal radiographs a diagnosis of protrusion of the disc can only be based on two findings, (1) a calcified protrusion into the vertebral canal and (2) diminished space between adjacent vertebrae. Of these criteria only the first is absolute proof of a protrusion, whereas the second finding is an indication that prolapse has occurred (Olsson, 1951a). To assist in estimating the spatial relationships Olsson (1951a) points out that as an approximation one may assume that a normal disc should not be narrower than the surrounding discs. Any interpretation of radiographs should, as always, take into account other clinical findings. Olsson (1951a) gives a warning that using this technique, a chronic protrusion that is not causing symptoms will often show on the radiograph and yet a symptom-producing prolapse will remain undetectable.

By using contrast media to outline the spinal cord the diagnosis of disc protrusions can be made more certain. Such an examination should be performed with the greatest care and preferably only when the dog is to undergo surgery (Olsson & Hansen, 1952). As the epidural space is filled with fat the obvious route is by sub-occipital puncture into the cisterna magna to show the sub-arachnoid space. Greene (1951), however, uses epidural injection between the last lumbar and first sacral vertebrae and operates on the strength of the resulting radiographs. Olsson (1951a) uses Thorotrast* as his contrast medium ; this is a colloidal preparation containing 24 to 26 per cent. thorium dioxide and miscible with body fluids, saline, etc. It may be toxic and it is advisable to drain off as much of the medium as possible after use. The smallest dogs require about 1 c.c. and the largest about 5 c.c. (Olsson, 1951a). More recently Olsson & Hansen (1952) have used such small doses of the contrast medium that drainage has been considered unnecessary. Contrast with this material is excellent. To drain off the fluid after use, ½ to 1 litre of hypotonic (0·45 per cent.) saline is injected intravenously at about 30 c.c. per minute ; the contrast medium and some cerebrospinal fluid will then flow back out of the needle which has been re-inserted in the cisterna magna (Olsson, 1951a). Cerebrospinal fluid drainage by the introduction of hypotonic saline solution is recognised clinically as a relatively safe method of irrigation of the sub-arachnoid space in man (Nosik & Mortenson, 1938). Hoerlein (1953) prefers to use Pantopaque* as a contrast medium ; this is an absorbable oil-type contrast medium of low viscosity and this worker reports that although no attempt has been made to drain it off after use adverse effects have not been noted.

After injection the dog is placed on a slope of 30° to 45°, tail low, for up to 20 minutes, being turned occasionally to promote even distribution of the medium. If Thorotrast has been used in anything but the smallest quantity the head of the animal must be on a pad to stop the medium entering the cranial cavity. Dorso-ventral, ventro-dorsal, lateral, and oblique views can then be taken, moving the head of the X-ray set rather than the dog.

The resulting radiographs should give a very clear idea of the condition of the sub-arachnoid space and even very small projections will cause some degree of curving of the membranes. Large projections may cause a complete block to the flow of the medium and, as some swelling of the tissues in the vertebral canal is not uncommon as a result of the prolapse, the contrast shadow may stop or thin out before the actual protrusion is reached (Olsson, 1951a). It must be borne in mind that all the radiograph can show is whether or not the sub-arachnoid space is still patent and if there are elevations in the floor of the spinal canal : even if a disc protrusion has caused an elevation or block it is not necessary that that obstruction is the cause of the symptoms. Interpretations of contrast radiographs should take into account the results of normal X-ray and neurological examination (Hoerlein, 1953).

Thorotrast-myelography is contra-indicated in shocked animals or when the general condition is poor (Olsson, 1951a).

Differential Diagnosis

According to Hoerlein (1953), infectious diseases (distemper, hard pad, etc.) and their sequelae may give rise to symptoms resembling those of disc prolapse. So also may tumours and abscesses of the cord itself and of the tissues surrounding the cord. Congenital abnormalities, fractures, and luxations, of the spine must also be borne in mind. Pachymeningitis ossificans and spondylitis (spondylosis) deformans are conditions occurring in old dogs which give rise to symptoms resembling dorsal disc protrusions. There are also some other conditions (*e.g.*, taeniasis (Hoerlein, 1953)) in which pain, paresis or paraplegia may occur but the history and other clinical findings should rule these out. Most of the other conditions mentioned can be dismissed when radiographs of the animal are available. Hoerlein (1953) describes a series of 79 cases which could be symptomatically confused with dorsal disc protrusion. Of these just one-half were proved by radiography or *post-mortem* examination to be due to disc prolapse. In a report on a series of *post-mortem* examinations of 273 dogs with a clinical history of disease of the central nervous system, McGrath (1951) groups 79 as showing paresis or paralysis of one or more extremities due to cord involvement : encephalomyelitis, which had frequently shown systemic symptoms, had accounted for 27 of these and of the remaining 52, 24 are grouped as intervertebral disc cases. This worker also has a " miscellaneous " group in which three additional cases " appeared to be (due to) protrusion of an intervertebral disc."

Post-mortem Findings

The neural arches are removed from all the vertebrae from the atlas to the sacrum. The spinal cord is carefully lifted out, starting at the atlanto-occipital junction, the spinal nerves

* Produced by Heyden Chemical Corp., N.Y., and imported by Savory & Moore.

* In this country Myodil (Glaxo Lab., Ltd.) is used in nearly all those radiography cases in which Pantopaque is used in U.S.A.

being cut at each intervertebral foramen. Care must be taken that the floor of the vertebral canal is not touched by instruments.

Discs.—As may be assumed from the section on the pathology of the disc the prolapses can take on several shapes, sizes and positions. A broad division may be made as to whether or not the annulus fibrosus has been completely ruptured.

Complete Rupture of the Annulus Fibrosus.—Acute cases of disc protrusion resulting in a progressive paralysis may only show a tear in the dorsal annulus with little or no prolapse material but with sometimes a marked epidural haemorrhage (Hoerlein, 1953). In many cases, however, the prolapse is of considerable size, irregular, brittle, and sometimes forming adhesions to the dura mater. A strong inflammatory reaction may have taken place in these instances, associated with hyperaemia, oedema, and some haemorrhage. When the acute inflammatory symptoms decrease, the colours fade from grey-red to grey-yellow and the consistency of the protrusion becomes firmer (Hansen, 1952). Hansen (1952) classifies this group as Type 1 prolapses and claims that a direct continuity between the prolapse and the inner degenerated parts of the disc can usually be traced. They are commonest in dogs of the chondrodystrophoid breeds (Hansen, 1952) and may occur to either side or even through the dorsal longitudinal ligaments (Figs. 4 and 5). In some cases, although there is a complete rupture of the annulus fibrosus, projection of prolapse material may remain under the dorsal longitudinal ligament (Hoerlein, 1953) (Fig. 3).

Incomplete Rupture of the Annulus Fibrosus.—These lesions correspond to Hansen's Type 2 protrusions (Hansen, 1952) and are characterised by a more limited regular shape, smaller size, firmer consistency, and usually a smooth even surface. They are formed by a bulging of the lamellae and the ligaments that have not yet been ruptured (Fig. 2). This type of prolapse occurs in old animals of all breeds and would appear to be a senile phenomenon.

Although it is more common to find a single disc protrusion this is far from the rule. Hansen (1952) examined 93 animals showing disc protrusions (not all of them clinical cases of the condition) and of these 39 showed single protrusions. Of the remainder, 16 had two protrusions, 17 had three, nine had four, five had five, three had six, two had seven, one had eight and one had nine.

Cord.—The lesions in the affected spinal cords have been dealt with briefly by Hoerlein (1953) and to a certain extent by Fankhauser (1948) and McGrath (1951). In acute cases the cord presents evidence of a severe concussion ; in more chronic cases the picture resembles that of a compression demyelination which may extend no farther than one vertebra either side of the lesion, being due mainly to the circulatory disturbances following compression of the blood and lymph vessels (Hoerlein, 1953).

Regarding the extra-dural condition of the canal, the amount of haemorrhage seems to be very variable and often some deposit and inflammatory tissue is present (Hoerlein, 1953).

Pathogenesis

The examination of factors influencing the development of a disease is necessary to appreciate the line of treatment and the prognosis. Olsson (1951a) conducts his investigations in three sections : (1) the factors determining the symptoms ; (2) the possible reasons for spontaneous healing, and (3) the causes of relapses. However, it is perhaps more convenient here to examine first those factors which cause a disc to rupture ; consideration of the three features which Olsson mentions will then follow.

The Circumstances Influencing Disc Protrusion.—Hansen (1952) devotes a large part of his monograph to this aspect of the disease and this is the source of the material for this section. It is convenient to examine the circumstances present in the two divisions of breeds.

Non-chondrodystrophoid Breeds.—As has been pointed out above (see Pathology of the Disc) a fibroid metamorphosis of the nucleus pulposus occurs in middle-aged to old dogs as a maturation phenomenon and may be associated with degenerative processes. In the annulus fibrosus degenerative changes occurring in the inter-lamellar zones are usually in the nature of a disintegration and loosening of the connection between the lamellae, associated with large or small foci of an indifferent granular mass. Lamellae may also show a hyaline degeneration and partial ruptures, the latter being most common in the dorsal part of the annulus. The dog may still have an apparently normal nucleus pulposus as degeneration can occur in the annulus fibrosus before any such change occurs elsewhere. We have, then, the " shock-absorbing centre " which may still be functionally uninjured roofed by lamellae, partly or completely ruptured or separated from each other. These are the conditions for the appearance of those lesions which Hansen terms " intra-discal protrusions " where some of the lamellae may be intact but subject to dorsal bulging. It is the type of protrusion most commonly found in dogs of the non-chondrodystrophoid breeds, usually after middle-age, and may occur when the animal is subjected to the normal stresses of life but is more liable to arise with greater strains.

Chondrodystrophoid Breeds.—In individuals of these breeds the mucoid nucleus pulposus has usually been replaced by chondroid tissue by one year of age. The nucleus pulposus has thus lost at an early stage its plasticity and hence its ability to function as a shock absorber. Before the central part has completely metamorphosed, more or less extensive degenerative changes with secondary calcification may have already appeared in the peripheral part of the nucleus pulposus and the peri-nuclear part of the annulus fibrosus. The central chondroid part of the nucleus pulposus is soon surrounded on all sides by a ring of necrosis and calcified material. Degenerative changes in the outer lamellar parts of the annulus fibrosus have not been observed without comparatively comprehensive changes in the central part of the disc. These lamellar changes resemble those occurring in other breeds. Material from the inner part of the disc may gradually force its way dorsally, the ventral part of the annulus fibrosus being thicker and less prone to ruptures. Although the chondrification of the nucleus pulposus is the start of the disease the end results will be determined largely by mechanical factors. In this condition of a non-plastic centre, peri-nuclear necrosis and/or calcification, and some degree of degeneration and rupture of the lamellae, normal movement may result in a traumatic effect on the disc. The effect of such conditions as forced restraint, pressing under gates, etc., will obviously be most severe.

What Factors Determine the Symptoms ?—Riser (1946) believes that the symptoms are determined by the location of the disc and the degree of compression of the spinal cord. Hoerlein (1953), as do all other workers, agrees with the first of these but believes that the degree of pathological response is the second important factor. This term is used to cover the individual variation in adaptation and response to compression of the cord. Olsson (1951a) is more concerned with the dynamic factor of the rate and method of development of the protrusion. To illustrate these features he draws attention to the slight symptoms that occur for a long time during the growth of a slowly enlarging intra-spinal tumour and yet the severe results that usually occur if the spinal cord is very carefully handled even for a very short time during an intra-spinal operation. Olsson also points out that pro-

trusions almost equal in size cause extremely varying symptoms, thus questioning whether size itself is of significance.

In describing cases of sudden paraplegia and death this worker believes that the nucleus pulposus empties explosively and yet no noticeable compression of the cord need be visible *post-mortem*. Obviously here a dynamic factor is at play. Can this same factor influence the more chronic cause of this disease? On this Olsson (1951a) says: "Via the rupture of the annulus fibrosus the protrusion is in contact with the nucleus pulposus. The altered pressure, which occurs within the disc when the animal moves and which is normally distributed over the discs, may then be transmitted to the protrusion and support an irritation of the nerves and spinal cord." It would seem, then, that the rate and method of development of the protrusion rather than the degree of compression of the cord influences symptoms. Hoerlein's observation should be borne in mind, that the adaptability and response of the cord to compression is also significant (Hoerlein, 1953).

The site of the protrusion must necessarily play some part in determining the symptoms. Symptoms from lesions occurring in the cervical region differ from those caused by lesions occurring in the thoraco-lumbar region, but lesions from adjacent discs can give rise to symptoms so similar that Hoerlein (1953) points out that even employing neurological techniques the precise localisation of the injury is most difficult. Whilst discussing the effect of the position of the protrusion it may be opportune to mention those cases of protrusions which very rarely cause symptoms. It is likely that small elevations will not produce symptoms noticeable to the owner (Olsson, 1951a). Olsson (1951a) describes the site of other non-symptom-producing prolapses as being between the sixth and seventh lumbar and first sacral vertebrae. Protrusions must be very large in this region to compress the cord, as the dural sac lifts from the floor beginning at the sixth lumbar vertebra.

Why does Spontaneous Healing Sometimes Occur?—According to Olsson (1951a) the ability to heal spontaneously will depend on two features, provided the change in the spinal cord is repairable. The compression may decrease or the cord may adapt itself to the new space. Fankhauser (1948) is of the opinion that the spinal cord is sometimes able to function quite well in spite of marked compression. Olsson (1951a) supports this opinion. Regarding the possibility of decrease of a protrusion this worker offers the following information. As the acute protrusion becomes chronic, so the inflammatory reaction diminishes; thus the compression should also decrease. The possibility of resorption of protruded tissue should also be borne in mind. Furthermore, by stabilisation of the protrusion the dynamic factor mentioned above will be negatived. For these various reasons the symptoms of cord irritation may abate.

Why do Relapses Occur?—An obvious cause of a relapse is the rupture of another disc. However, re-rupture of a healed lesion does sometimes occur and also sudden increase in size of an existing prolapse can initiate another train of symptoms (Olsson, 1951a).

Prognosis

Riser (1946) suggests that prognosis should depend on (1) the number, type and extent of protrusions in the canal, (2) the extent of damage to the cord, (3) the ability of the disc and cord to repair, and (4) the influence and extent of secondary infections and the ability of the animal to adjust itself to its new state. Of these factors the first is the only one on which definite information can be gained and that by radiography. Olsson (1951a), however, believes the dynamic effect of a protrusion rather than its topography to be of importance. It would seem that even the little definite information we can gain is of limited prognostic value. Should the other factors enumerated by Riser, which cannot be accurately assessed, appear to be favourable and if stabilisation of the protrusion can be achieved (see Treatment) then the short-term prognosis is good. Bearing in mind the condition of all the discs in the spine (as outlined in Pathogenesis) it is apparent that further prolapses can occur, and this likelihood must be evaluated in prognosing cases of disc protrusion.

Treatment

Two lines of treatment are now open, medical and surgical.

Medical Treatment

Natural healing is encouraged by complete rest, sometimes with the aid of sedatives. As far as we are aware, no one has assessed the relative merits of various drugs in the different symptom complexes. As vitamin B_1 can contribute to the improved metabolism of nervous tissue this line of therapy is often instituted in the hope that the damaged nerve tissue will repair more quickly. Careful nursing and the avoidance or symptomatic treatment of secondary infections are obvious measures to be applied (Olsson 1951a).

Surgical Treatment

Two techniques have been employed in an effort to influence the condition favourably. In the operation of laminectomy an attempt is made to remove the protrusion, whereas in the disc fenestration operation the intention is to "unload the pressure in the disc" (Olsson, 1951a).

Laminectomy.—This operation has been described by Greene (1951), Redding (1951) and Olsson (1951a). Greene (1951) conducted his operations on cases showing lesions due to disc protrusions, but does not give the number of cases or mention results, except to state: "No improvement should be expected in less than 30 days. Best results are obtained in those cases which are diagnosed and operated on at the earliest possible moment, since the damage to the cord is proportional to the age and extent of the injury."

Redding (1951) performed laminectomy on 10 normal dogs without serious sequelae in nine, and concludes that "results indicate that the operation might offer a satisfactory approach in the surgical treatment of protruding intervertebral discs." Olsson (1951a) operated on five dogs, three of which were clinical cases. The two normals were very slightly affected by the operation. Two of the affected ones, which had pain and slight paresis pre-operatively, had paraplegia post-operatively but regained a certain faculty of locomotion after about two months; the pain syndrome had vanished. The fifth case was a paraplegic which improved to some extent after two months.

Technique.—The skin is incised over the area above the right or left border of the dorsal spines of the vertebrae involved. Fascia and muscles are reflected from the vertebrae, taking care not to damage spinal nerves in the area. The neural arch of one side with attached facets is removed either by rongeur or trephine. If the protrusion can be seen it is removed, avoiding if possible touching the spinal cord. The wound is closed in the usual manner.

Disc Fenestration.—The rationale for this treatment is explained by Olsson (1951a) as follows: "Via the rupture of the annulus fibrosus the protrusion is communicated with the nucleus pulposus. By body motion the nucleus pulposus is exposed to pressure which is transmitted mainly to the protrusion via the rupture instead of being evenly distributed over the whole disc. To fenestrate means here to make an incision in the annulus fibrosus into the nucleus pulposus (from) outside the vertebral canal. The pressure on the

protrusion should then decrease because of most of the pressure being conducted through the fenestra. In cases of incomplete protrusions with only a bulge of the outer layers of the annulus fibrosus this bulge should decrease."

In the paper presented at the International Veterinary Congress, Hansen & Olsson (1953) suggest that dogs with cervical disc lesions and those with slight or medium symptoms of long standing should be operated on. If such symptoms are acute, the dogs may be operated on ; this will reduce the risk of a prolapse increasing in size and, if adjoining discs are also fenestrated, of another prolapse occurring. They recommend that operation should not be attempted if (1) radiographs show a large number of calcified discs all along the spine, (2) the dog shows grave symptoms, (3) the dog is shocked or in bad general condition.

Olsson & Hansen (1952) report that 57 dogs with thoraco-lumbar disc protrusions were operated on in the years 1950 and 1951. By 1952, 10 cases had relapsed ; five of these were available for re-examination and showed the relapses to be due to protrusions of non-fenestrated discs. Four cases of cervical disc protrusions, surgically treated, completely recovered.

Technique.—(Olsson, 1951a, and Olsson & Hansen, 1952.) Two to three days are allowed to elapse after myelography to allow the reaction to wane. For thoraco-lumbar intervention the skin and fascia are incised alongside the spinous processes. The muscles are reflected down to the transverse processes after stripping them from the vertebrae, taking care not to damage nerves and vessels in the vicinity. To operate on cervical discs the approach is via a skin incision along the trachea ; the disc can then be reached by using only blunt instruments. To operate on thoracic discs cranial to thoracic vertebra 12 the approach is transthoracic by removal of a rib and the employment of artificial respiration. The disc in question is then fenestrated dorso-laterally (ventrally in the case of cervical discs) with a small knife or lancet. As much as possible of the loose content of the disc is removed with a scoop. The neighbouring discs can easily be fenestrated through the same opening ; this may be an advisable prophylactic measure. The wound is closed normally.

PART III.—CASE REPORTS

The following two case histories are representative of our trial series of seven cases of locomotory paralysis in which the cause was attributed to disc protrusion. It is, of course, practically impossible in any given case to prove the absence of distemper and distemper-like diseases and gross trauma. However, the practitioners who were in charge of these cases, and we ourselves, were satisfied from the history that these conditions could be excluded.

Case No. 1

Clinical History.—The animal was a vigorous, four-year-old, male cocker spaniel. He had been in perfect health and under constant observation.

There was an initial period for about six hours of sluggishness and unwillingness to move. The onset of acute symptoms was sudden while out walking ; he leapt on to a wall, and instantly cried out. He was immediately unwilling, or unable, to stand and walk and had to be carried home. The hind limbs were certainly totally paralysed on arriving home some three minutes later. From this time until examination 48 hours later the symptoms were paralysis of the hind limbs, repeated bouts of crying out, resentment of handling, attempts at turning and biting at the back, dribbling of urine and cessation of defaecation.

On examination there was flaccid paralysis of the hind limbs with occasional clonic spasms of the forelimbs. The patella reflex was exaggerated. Pricking of the skin revealed an apparently reduced sensitivity over the abdominal wall and hind limbs ; the response to manipulation suggested hyperaesthesia of the late thoracic and early lumbar vertebral areas. A second examination 60 hours after onset showed a general deterioration with little response to external stimuli. Respiration had become rapid and very distressed. Apart from the dyspnoea and occasional crying out the body was inert. Collapse seemed to be impending. Destruction was carried out.

Post-mortem Examination.—The neural canal was opened dorsally from the mid-thoracic to the sacral vertebrae. The epidural fat was haemorrhagic, markedly so at the thoraco-lumbar junction. The cord was much discoloured with subdural haemorrhage as far as it was exposed.

Over the articulation of the last thoracic and first lumbar vertebrae the dorsal longitudinal ligament, which was slightly elevated by a pale swelling, was split longitudinally in the mid-line for 4 mm. (Plate I, Nos. 2 and 3). A low ridge of yellowish material protruded from the whole length of this split ; in addition two ragged tufts (diagonally hatched in Plate I, No. 3) projected for 2 to 3 mm. from the centre, one on each side of the mid-line. The cord was inspected especially at the site of protrusion. Here, in a dorsal and lateral area about 1 cm. long, the subdural haemorrhage was not more marked but was different in colour, being pink rather than dark blue as elsewhere. However, the precise part of the cord which had been in contact with the protrusion was comparatively free of haemorrhage.

Discussion of Case No. 1.—Spaniels are not included in the chondrodystrophoid group of breeds. However, Hansen (1952) has shown that the breed has chondrodystrophoid tendencies (see Pathology of Disc) ; moreover Hoerlein (1953) has found a higher incidence of protrusion than in the true

Plate I.—Four photographs of Case No. 1. The photograph of the vertebral column is reduced to approximately half life size ; the enlargements are approximately twice life size. A line connects each enlargement to the level of the appropriate intervertebral region on the low-power photograph.

No. 1, dorsal view of late thoracic and lumbar vertebrae with vertebral arches and contents of canal removed to show floor of canal. Protrusion of disc material at junction of 13th thoracic and 1st lumbar vertebrae, other discs normal.

No. 2, enlargement of this lesion, showing a bulge under the dorsal longitudinal ligament with a small median split and material protruding through it.

No. 3, as No. 2, but protruding material is outlined in ink ; a low ridge protrudes all along the split and two tufts (hatched) project from the centre.

No. 4, a normal for comparison.

Plate II.—Nine photographs of Case No. 2. Magnifications and directions as for Plate I.

No. 1, dorsal view of late thoracic and lumbar vertebrae with vertebral arches and contents of canal removed to show floor of canal. Six intervertebral areas are abnormal and are shown as enlargements, with one normal for comparison.

No. 2, 11th to 12th thoracic intervertebral space with a hard gritty projection through the dorsal longitudinal ligament to the left of the mid-line.

No. 3, a small oblong piece of dura mater lying parallel to the dorsal longitudinal ligament remains attached to the floor of the canal on the right of the mid-line and just cranial to the 12th to 13th thoracic intervertebral space.

No. 4, normal 13th thoracic to 1st lumbar intervertebral space.

No. 5, shows a knob-like protrusion splitting the dorsal longitudinal ligament to the left of the mid-line over the 1st to 2nd lumbar intervertebral disc.

No. 6, a hard white swelling overlies the 3rd lumbar vertebra.

No. 7, a mid-line tear over the 4th to 5th lumbar intervertebral space with some protruding material.

No. 8, a mid-line tear about 1 cm. long over 5th to 6th lumbar intervertebral space with some protruding material.

No. 9, as No. 8, but protruding material and split outlined in ink.

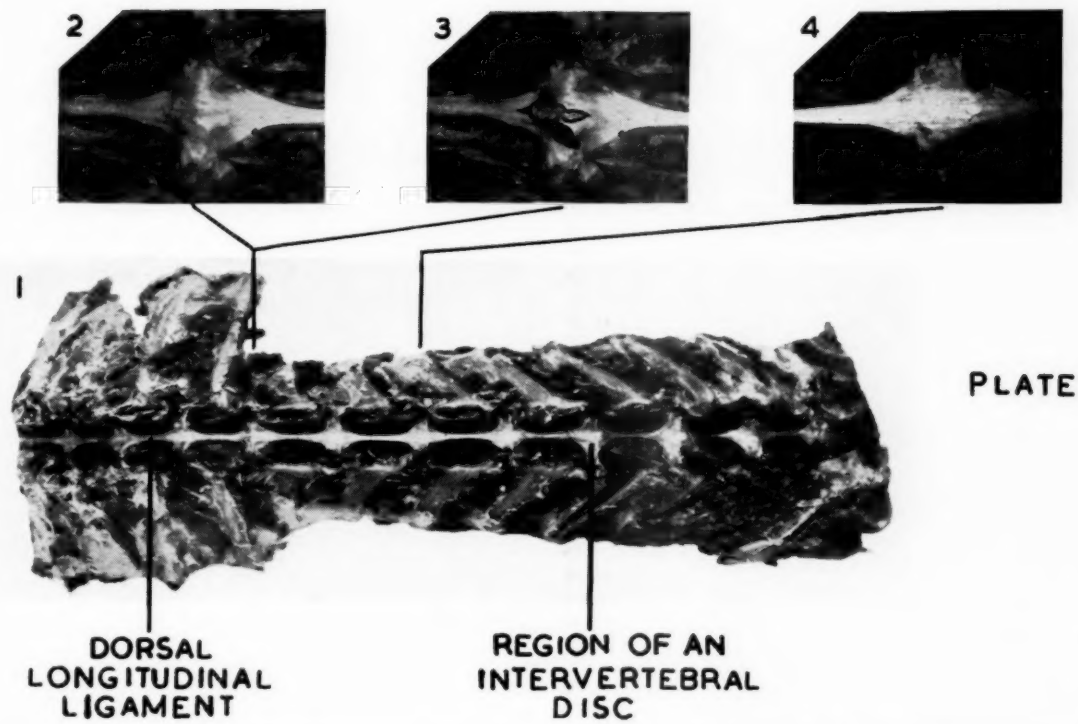

PLATE I

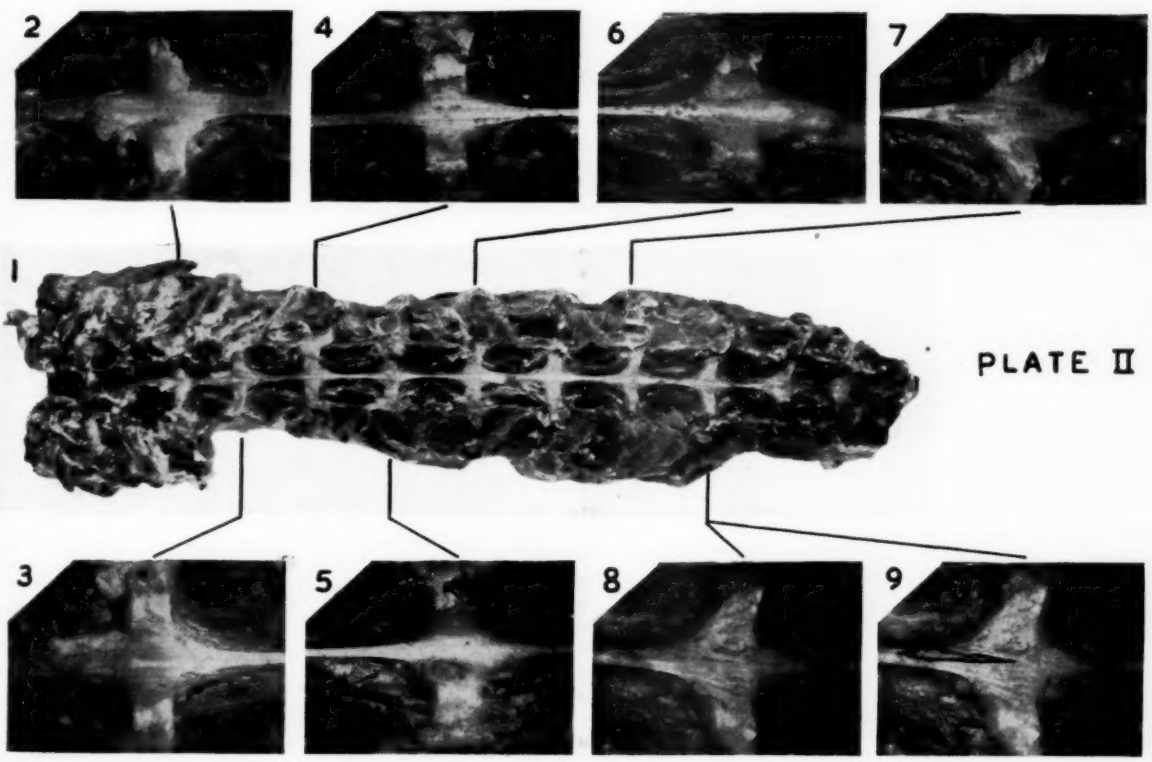

PLATE II

non-chondrodystrophoid breeds. The symptoms are typical of those described by the main authorities for acute disc protrusion ; they resemble case histories published by Olsson (1951a) and Hansen (1952) and also conform to the main characteristics of progressive ascending paralysis (syndrome 4 : see Symptoms) as summarised by Hoerlein (1953), *i.e.*, a sudden physical strain followed abruptly by pain, severe sensory and motor paralysis of the hind limbs, and dysfunction of the anal and urinary sphincters, with progressive deterioration. The striking respiratory changes are regarded by Riser (1946) as characteristic. The *post-mortem* findings are also typical of acute disc protrusion in their resemblance to the features listed by Hoerlein (1953) ; *i.e.*, rupture of the dorsal longitudinal ligament, extensive subdural haemorrhage, and reddening of the epidural fat. The protrusion itself is a complete dorsal median one with splitting of the dorsal longitudinal ligament, as shown in Fig. 5.

Case No. 2

Clinical History.—The notes for this case are taken from the veterinary surgeon's day cards, which were kept more for administrative than for clinical purposes. The subject was a seven-year-old male dachshund which four years ago had become posteriorly paralysed. Heat treatment was recommended and in about a month the dog had returned to normal. Eight months after the initial attack there was a bout of rheumatoid pains of a week's duration which were symptomatically treated. These symptoms reappeared four months later, and very shortly were followed by posterior paresis. Various treatments were given and the animal was then subsequently free from locomotory disturbance for a year, during which time he was treated for kidney trouble. Two years ago he was spontaneously and completely paralysed in the hind region. Gradual improvement took place and after a fortnight he was able to walk again. He was unable to empty the bladder or expel faeces and for about 12 months these actions were aided manually each day. After this longer periods were instituted for the treatment. He was destroyed in May of this year because of a recurrence of the kidney trouble in a more severe form.

Post-mortem Examination.—There was no haemorrhage in the spinal canal, but the floor showed many lesions. Just anterior to the 11th to 12th thoracic intervertebral space was a hard gritty projection of about 2 cu. mm. protruding through the dorsal longitudinal ligament to the left of the mid-line (Plate II, No. 2). The dura was firmly attached to the ligament to the right of the mid-line and just cranial to the 12th to 13th thoracic intervertebral space over an area 5 mm. long and 2 mm. wide (Plate II, No. 3). The 13th thoracic to 1st lumbar space was normal (Plate II, No. 4). To the left of the mid-line and immediately over the 1st to 2nd lumbar intervertebral space a protrusion 4 mm. long, 2 mm. wide and 1 mm. high was splitting the dorsal longitudinal ligament (Plate II, No. 5). Over the third lumbar vertebra a hard white swelling of the ligament, 5 mm. long and 2 mm. high, occurred in the mid-line (Plate II, No. 6). The 3rd to 4th lumbar intervertebral space was normal. Over the 4th to 5th lumbar intervertebral space was a mid-line tear with some protruding material (Plate II, No. 7), and over the 5th to 6th space was a longer tear, also with some protruding material, of about 1 cm. length (Plate II, Nos. 8 and 9).

Discussion of Case No. 2.—The course of this case is typical in that it shows acute symptoms starting in a young dog of a chondrodystrophoid breed. The ensuing history of spontaneous healing with relapses is also a clinical feature of the disease. The period of rheumatoid pains is interesting in view of Olsson's contention (1951a), mentioned in the section on symptoms, that those pain symptoms which give rise to a diagnosis of rheumatism and myalgia should be considered as due to disc protrusions. The *post-mortem* findings bear out the record of the multiplicity of attacks although it is extremely difficult to attempt to correlate a specific bout of symptoms with any individual lesion. Plate II, Nos. 2, 5, 7 and 8, are lesions similar to that shown in Fig. 5, whereas No. 6 might resemble either Fig. 2 or Fig. 3.

PART IV.—DISCUSSION

Although so much has been written in other countries, protrusion of the intervertebral disc has apparently not been fully accepted in this country as a major cause of locomotory paralysis in the dog. (For a brief review of British reactions see Part I, Introduction.) The observer who comes fresh to the topic will therefore wish to inspect critically the results published abroad. The following discussion attempts to expose impartially the strength and weakness of this evidence. Personal opinions are occasionally given in summary, but will not hinder independent judgment by others.

To show beyond reasonable doubt that protrusion of the intervertebral disc is a major cause of locomotory paralysis in the dog, at least two main requirements must be satisfied. It must be shown, first, that the nervous symptoms are closely associated with projections into the neural canal and secondly, that these projections are derived from an intervertebral disc. Two sections are now devoted to assembling the evidence relevant to each topic.

Is There a Close Association between Nervous Symptoms and Projections into the Neural Canal?—The first essential is a combined clinical and morbid anatomical investigation of a sufficiently large number of cases. Every case should have as its main symptoms those regarded as typical by the principal authorities (Hoerlein, Hansen and Olsson), namely, pain, locomotory paresis and paraplegia. It would need to be shown that in a substantial proportion of these cases there was a swelling projecting into the neural canal. An investigation of approximately this type was undertaken by Hoerlein (1953) : of 79 cases with these symptoms the presence of protrusions into the neural canal was proved to his satisfaction in 41 ; a definite alternative diagnosis was found for the majority of the remaining 38. The main defects are these. First, the total number of cases is somewhat small. Secondly, in the 41 positive cases, the presence of a projection was in some instances (the number not being given) deduced from radiographs alone and not from *post-mortem* examination : however, the experience of the present authors has convinced them that an error from this source is unlikely to be great. Thirdly, Hoerlein did not personally examine all the cases.

It is now necessary to determine whether or not the projections are likely to have caused the nervous symptoms. Three features of the projections must be analysed, the site, size and texture. A great deal of evidence is available on the site since the Swedish contributions also can now be included. Cases of paraplegia have been consistently found with projections in the lumbar region, and cases of forelimb paralysis with projections in the cervical region. Comparable relationships between sensory changes and the location of projections have also often been shown (Hoerlein, 1953). So far the association between the main symptoms and the projections appears remarkably close, and the strength of this evidence should receive due emphasis. On the other hand, a more detailed neurological analysis of the association is less assuring. Why should a projection at the thoraco-lumbar junction cause acute respiratory distress ? Why, indeed, should some cases with lumbar projections be quite rapidly fatal ? However, neurological interpretations are intrinsically so difficult that failure to explain everything is not a vital defect. The evidence on the size and texture of the swellings

is less satisfactory. In some of the photographs published by Hansen (1952) and in one of our own cases (see Plate II, No. 5) there are swellings beneath the dorsal longitudinal ligament which are quite obviously large enough to compress the spinal cord. Moreover, the texture of this sort of swelling is typically firm and hard. However, a substantial proportion of the projections found at *post-mortem* examinations of clinical cases seem to be so small that the normal mobility of the cord should allow it to escape the pressure. Particularly difficult are the cases (for example, Case No. 1 of our own) where there is no marked swelling beneath the dorsal longitudinal ligament but predominantly an escape and spreading of disc material into the neural canal (see Plate I, Nos. 2 and 3). Olsson (1951a) offers one explanation of how these latter protrusions may cause symptoms, by suggesting that the liberation of material into the neural canal occurs with "explosive" violence. It is not easy to accept an explanation essentially so theoretical and undemonstrable. On the other hand, it may be significant that, as Hoerlein (1953) has shown, these protrusions are in fact typical of the *acute* cases with sudden dramatic symptoms which sometimes even culminate in death (our own Case No. 1 being an example).

Histopathological evidence is also relevant. If there is a direct association between the symptoms and protrusions it should be possible to demonstrate lesions in the spinal cord in the immediate vicinity of the projections. Hoerlein (1953) has made histopathological examinations of the cord in chronic and acute cases. In the former he found comparatively mild lesions which were indeed "in most cases . . . quite local" extending "no more than one vertebra to either side of the lesion" (protrusion). He appears to regard these lesions as compatible with compression of the cord. In acute cases he found severe lesions but unfortunately does not say whether they were topographically related to the protrusions; however, he does interpret the picture as being "largely one of severe concussion."

Finally, if the protrusion causes the symptoms, removal of it should relieve them. Greene (1951) and Olsson (1951a) have tried to do this by the direct operation of laminectomy. But the results, though mainly favourable, are far from decisive. Olsson (1951a) used the indirect method of fenestration, which is designed to release the pressure inside the affected disc. He expects that an incomplete protrusion, with the annulus fibrosus still intact, should then decrease; at the least he anticipates that further advances of the protrusion, which he believes may play an active part in inducing symptoms, should be prevented. He carried out the operation on 28 dogs* which he divided into two groups, one with severe symptoms (paresis and paraplegia dominating), the other with milder symptoms (pain, with less prominent motor effects). Of the 13 dogs in the latter group 11 recovered fully, one partly, and one died "on account of the operation." Olsson further commented: "It is very remarkable, however, that eight of these dogs did not only recover, after operation, but also regained a vitality not seen since long before their owners had noticed the disease." Of the 15 dogs in the acute group 12 recovered to the extent that "only a trained observer could notice slight ataxia," one recovered partly, one was killed because there was no improvement, and one failed to improve and died from other causes. The significance of these results depends on comparison with the results of giving no specific treatment. Olsson applied a comparison to 21 dogs with treatment based on rest and injections of vitamin B_1. The animals were divided into the same two groups. Of the 10 dogs in the mild group five recovered fully, two partly, and three were killed because there was no improvement. Of the 11 in the severe group five recovered fully, three partly, and three were killed because they failed to improve. The substantially higher incidence of full recovery and lower incidence of complete failure after fenestration seems to provide rather strong evidence of a direct connection between the symptoms and the protrusions. However, the number both of fenestrated and non-fenestrated dogs is small.

The evidence on the possible association between symptoms and projections can now be summed up. First, a combined clinical and morbid anatomical investigation has shown that of 79 cases of paresis, pain and paraplegia, 41 had projections into the neural canal. Secondly, the site of the projections conforms to the main symptoms: sometimes also the protrusions are sufficiently large and hard overtly to cause pressure on the cord, and on other occasions there is a possibility that their liberation is violent. Thirdly, the histopathology of the spinal cord shows, in chronic cases, lesions closely localised to the site of the protrusions, and, in acute cases, lesions indicating severe concussion. Fourthly, there is surgical evidence that reduction and/or stabilisation of the protrusion relieves the symptoms. But, no single piece of this evidence is perfectly sound.

In short, the items of evidence agree consistently but are individually deficient. To give a personal view the present authors think that the degree of agreement compensates for the deficiencies, and thereby permits the belief that in a substantial proportion, possibly even the major proportion, of cases of paresis, pain and paraplegia, the symptoms arise from projections from the floor of the neural canal.

Are the Protrusions from the Floor of the Neural Canal Derived from Intervertebral Discs?—First, are the protrusions intervertebral in position? Fortunately there is abundant evidence, particularly from the Swedish work. The great majority of the protrusions overlie an intervertebral space.

Secondly, does the normal anatomy of the intervertebral disc offer opportunity for protrusion? Two features are relevant. First, the normal disc has a soft centre, the nucleus pulposus, which is dorsally excentric; thus the normal anatomy could provide both the missile and the route. Secondly, the strong conjugal ligament covers the intervertebral area from the second to at least the ninth rib, and might be expected to bar the route; Hoerlein (1953) found only a very small number of protrusions in these regions, and Hansen (1952) not one.

Thirdly, is there direct morbid anatomical evidence that the protrusions are derived in the manner suggested by the normal structures? From the examination of hundreds of protrusions Hoerlein (1953) and Hansen (1952) have revealed that there is usually direct continuity between the protrusion and the nucleus pulposus. This observation has also been made on some of our cases.

Fourthly, is there evidence from the morbid anatomy and histopathology to show why disc protrusion should occur? On a total of over 500 dogs Hansen (1952) examined macroscopically many hundreds of discs and made an extensive histological survey. In those breeds in which the protrusions (and the symptoms) are commonest, *i.e.*, the three chondrodystrophoid breeds, dachshund, French bulldog and Pekinese, he found initial chondroid changes in the nucleus pulposus in the majority of one-year-old dogs; from the age of two years there was calcification in over 50 per cent. of the discs, affecting mainly the nucleus pulposus but also the inner layer of the annulus fibrosus. In other breeds, in contrast, initial fibroid changes were not found with equivalent frequency until seven years of age, while degenerations were usually not seen until even greater ages and calcification was very seldom

* Although there is a subsequent report on 57 dogs (Hansen & Olsson, 1953) (see Treatment), the information given on the results is not so complete, which renders them unsuitable for the present discussion.

ound at any age. These results suggest that, in marked contrast to the non-chondrodystrophoid breeds, throughout most of the life of the chondrodystrophoid animal the majority of the intervertebral discs have degenerated : their centre has changed from resilient softness to intractable hardness ; the originally tough fibrous periphery of the nucleus pulposus is now brittle with calcification. It is difficult to escape the conviction that these results of Hansen demonstrate the " discogenic " nature of the protrusions. Nevertheless, it must be remembered that they have yet to be confirmed by other workers.

To sum up this section, the evidence is in close agreement and individually most of it has no serious blemish. The normal anatomy is well known. The morbid anatomy of the protrusions and of discs in general has been investigated on a large scale in Sweden and the former has also been confirmed by Hoerlein. The histopathology lacks confirmation, but otherwise appears entirely satisfactory. The present authors are compelled to the conclusion that the very great majority of the protrusions into the neural canal of the dog are derived from intervertebral discs.

Summary of the Discussion

An analysis has been made of the evidence, first showing the extent to which symptoms in the dog of locomotory paresis, pain and paraplegia may be attributed to projections into the neural canal, and secondly showing whether these projections may be derived from the intervertebral discs. The evidence on the first point is regarded as consistently in agreement but individually deficient ; it is thought to permit the belief that in a substantial proportion, possibly the major proportion, of cases the symptoms arise from projections into the neural canal. The evidence on the second point is also consistently in agreement and in addition is mostly free of individual weaknesses ; it is thought to compel the conclusion that the very great majority of projections into the neural canal of the dog are derived from intervertebral discs.

Finally, it is necessary to return to the original problem. Has it been shown beyond reasonable doubt that protrusion of the intervertebral disc is a major cause of locomotory paralysis in the dog ? Certainly it has not been proved. Before that could be allowed, various shortcomings, mainly in the symptomatological-morbid anatomical evidence, must be overcome. The prime need appears to be for a clinical survey, with *post-mortem* and/or X-ray examination, on a statistically satisfactory number of dogs showing locomotory paresis and paraplegia : this would test and amplify Hoerlein's valuable results. The monumental work of Hansen and Olsson, to which too great a tribute cannot be paid, may well already contain the information required. Both of them plainly regard the " discogen " nature of the disease as already beyond doubt ; consequently neither of them has published, as far as we know, an analysis of his results in a form applicable to proving a relationship between disc protrusion and paralysis. It would be most illuminating to see a new article from that source aimed solely at demonstrating once and for all " the discogen nature of the disease." Meanwhile, whether at present it is beyond reasonable doubt is a matter of opinion. In our view, because of the consistent agreement of the wide array of evidence and the individual strength of it in many places, it is.

PART V.—SUMMARY

1. The main landmarks in the literature on the condition now referred to as disc protrusion in the dog are reviewed. Although in 1896 Dexler attributed paraplegia to disc disease, Tillmans in 1989 was the first to suggest that the cause was prolapsed disc material. The main works on the topic are by Hansen and Olsson (1951 to 1953) in Sweden, and by Riser (1946) and Hoerlein (1953) in America. The only article in the British Press specifically on disc protrusion as a cause of canine paralysis appears to be that by Kirk in 1953.

2. From the above works and other articles an account of the disease has been extracted. The initial section is on the normal anatomy of the intervertebral disc. The pathology of the disc is reviewed with particular attention to age and breed factors ; these are examined through the changes in the disc peculiar to the chondrodystrophoid breeds, *i.e.*, dachshund, Pekinese and French bulldog. Symptomatology is reviewed on the basis of four syndromes, pain, paresis, paraplegia and acute ascending paralysis. In a section on diagnosis the rôles of normal and contrast radiography are considered. The differential diagnosis is outlined. The section on *post-mortem* findings includes the pathology of the cord, and the variations in disc protrusions dependent on whether the annulus fibrosus is partly or wholly ruptured. In the pathogenesis the mechanism of protrusion and of the development of symptoms is examined. Finally, medical treatment and the surgery of laminectomy and fenestration are reviewed.

3. Clinical and *post-mortem* findings are given of two cases selected as representative of our trial series of seven dogs showing locomotory paralysis but considered as free from distemper and other comparable diseases and trauma. The findings are regarded as typical of disc protrusion.

4. The evidence is discussed for protrusion of the intervertebral disc as a major cause of locomotory paralysis in the dog. First has been considered the extent to which symptoms in the dog of pain, locomotory paresis and paraplegia may be attributed to projections into the neural canal. The evidence is found to be consistently in agreement but individually deficient ; it is thought to permit the belief that in a substantial proportion, possibly the major proportion, of cases the symptoms arise from projections into the neural canal. Secondly, it is considered whether these projections may be derived from the intervertebral discs. The evidence is found to be consistently in agreement and is mostly free of individual weaknesses ; it is thought to compel the conclusion that the very great majority of projections into the neural canal of the dog are derived from intervertebral discs.

5. It is concluded that, although the available evidence does not amount to proof, it does show beyond reasonable doubt that protrusion of the intervertebral disc is a major cause of locomotory paralysis in the dog.

Acknowledgments.—We would thank Professor Ottaway for the interest he has shown in this venture. We wish to take this opportunity to thank the veterinary surgeons practising in Bristol for the great help they have accorded this Department since its inception. With regard to the clinical material referred to above, we would especially thank Mr. J. Allcock and Miss B. Penhale. The technical staff of the Department has continually rendered valuable assistance. For the photographs we are indebted to Mr. A. Coombs and to the Department of Medical Photography of the University.

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(*Concluded at foot of col.* 1, *page* 12.)

DANISH VETERINARY PRACTICE*

BY

JOHN B. EDWARDS, M.R.C.V.S.

WELLINGBOROUGH

As a result of an advertisement which appeared in *The Veterinary Record* some 18 months ago I got into touch with a Danish veterinary surgeon practising near the west coast of Jutland, and the following impressions and comparisons are the result of an exchange of visits between this Danish colleague and myself and also of a series of letters which passed between us.

The first thing on which the members of the Danish Veterinary Profession are entitled to our congratulations is the possession of a distinctive title. They are known not as "Vet." or Doctor but as DYRLAEGE, pronounced Durelayger, and are addressed as Dyrlaege Anderson, etc. I might add that this title even extends to their wives, who describe themselves as Frau Dyrlaege Anderson, etc. The title means literally "animal healer."

There is no equivalent to the Veterinary Surgeons Act, 1948, in Denmark, but there are in fact no unqualified practitioners. Certain farmers, however, may have a reputation for calving cows and this leads so often, as elsewhere, to a more difficult case when the qualified practitioner is eventually called in. The only legal control is the Cruelty to Animals Act which is very comprehensive and prohibits, for example, the use of any form of mechanical forced traction to deliver a calf or foal.

The general run of cases met in practice are essentially the same as in this country though there are still many more horses employed in agriculture than here. I propose, therefore, to confine my remarks rather to the organisation than to the technical problems of practice.

"*Ministry Work.*"—The first thing which struck me was the difference in Ministry work as we know it. The only duties carried out for the Ministry of Agriculture are those dealing with the control of notifiable diseases. The chief of these is the control of foot-and-mouth disease and I will try to give a typical instance. It will be apparent that the L.V.I. carries out the whole of the duties and does not hand over to a whole-time staff as in this country.

The owner of a suspected animal informs his V.S., who at once goes to the farm, leaves his car on the road, dons protective clothing and examines the affected animal, which, if positive, is at once removed from the shed. He then thoroughly disinfects himself, puts up notices of the existence of disease on the farm gates and doors, and informs the police and the Veterinary Inspector for contagious diseases for Jutland. The V.I. for Jutland can, if he wishes, enforce vaccination on farms within 2 km. of the affected place and on all farms supplying milk to dairies which also receive milk from the infected district. The police post notices around a 2 km. area from the infected farm, which remain in force for six weeks.

If the farmer wishes to use serum the usual type (A5) is used and saliva from the infected animal is scratched on to the tongue at the same time. The V.S. visits the farm again 18 to 21 days later and if all cases have recovered he arranges disinfection. He then returns when this process is completed to make sure it has been satisfactorily carried out. If so, eight days later restrictions are removed.

The farmer receives compensation for the loss of sale of milk only. This has to be weighed daily and is then boiled and fed to stock.

Tracing is done by the V.S. in charge of the outbreak without further reference to the Chief Veterinary Inspector unless this involves a great distance.

Payment for these duties is very low but the system, being complicated, is difficult to compare.

Vaccine used is produced from cows on the island of Lindholme. Cows are infected by virus injected all over the tongue (under anaesthesia); the cows are subsequently killed, tongues removed, tissue fluids pressed out and a vaccine prepared and treated with aluminium hydroxide.

Meat Inspection.—Throughout the country meat inspection is carried out by the Veterinary Profession.

In large towns there are abattoirs which are State inspected. The Supervisor in charge is responsible for the inspection of all meat, and either he or the Veterinary Inspector must be present at the actual slaughter. All animals are inspected before slaughter and all casualty carcases must be examined bacteriologically, except in cases of fractures.

In small towns the local V.S. is informed by the butcher when he has an animal for slaughter. It is inspected, marked with an ear tag and the carcase inspected after slaughter. The butcher pays the fee for this inspection direct to the V.S.

In the case of a casualty animal the V.S. can condemn the carcase but cannot pass it without first sending a kidney, piece of liver, gall bladder, portion of muscle and spleen with two carcase lymph glands to a laboratory and receiving its report.

Brucellosis and Tuberculosis.—I have put these diseases in order of relative importance to the Danes.

In the case of registered Brucella-free herds, milk is tested three times yearly at the dairy, a preliminary blood test being carried out before registration.

Where there have been reactors the herds are blood-tested every six months, reactors being slaughtered and in some cases Strain 19 is used on young stock.

Tuberculin testing is carried out every two years and is arranged and paid for by the Farmers' Co-operative Dairies. The test used is the mammalian only, any doubtful animals being retested by the comparative method.

Artificial Insemination.—This is no longer completely controlled by the Veterinary Profession and is carried out by laymen. Some V.S.s, at the request of the farmer, obtain the semen from the centres and carry out the insemination personally. The semen is sent out in small plastic tubes which fit on to a catheter. The instrument has a plunger which pushes the contents out of the plastic tube. The plastic tube is then discarded.

* An address to the Society of Practising Veterinary Surgeons, at Shrewsbury, on September 9th, 1953.

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System of Payment.—There is a scale of minimum fees issued by the Danish Veterinary Association which is circulated to its members. This scale is very detailed and covers every possible case which may be met in practice.

The fees for consultation and mileage are quite adequate and are intended to provide the V.S.'s income as there is only a small margin of profit allowed on vaccines, sera and drugs. It is interesting to note that there is an increase of 25 per cent. in fees after 6 p.m. and a further increase to 50 per cent. between 9 p.m. and 7 a.m.

The prices of drugs are fixed by law. The V.S. gets a discount and is allowed to add a small dispensing fee, varying according to the work involved, *i.e.*, whether solutions or compounding, etc., are required.

Those vaccines which are produced by the State Laboratory are supplied direct to the veterinary surgeon, who must charge a fixed price for them to his clients. In the case of foot-and-mouth vaccine the vaccination is usually by arrangement with the Farmers' Co-operative Dairy. The dairy purchases vaccine on behalf of its members and the V.S. is paid a small sum per head for inoculating. Those vaccines not produced by the State Laboratory are supplied through the chemists to the farmers, the V.S. being again paid for inoculating and also getting a small percentage from the chemist.

There are, I understand, no proprietary medicine vendors or " Veterinary Chemists " in Denmark.

In conclusion, I should like publicly to express my thanks to my great friend Dyrlaege J. C. Christensen and to all the Danes whom I met over there for the hospitality, information and advice which they so readily gave and their many expressions of friendship to this country.

REVIEW

[M'Fadyean's Osteology and Arthrology of the Domesticated Animals. Fourth edition (1953) edited by H. V. Hughes, B.V.Sc., M.R.C.V.S., D.V.H., and J. W. Dransfield, M.V.Sc., M.R.C.V.S. Pp. 288, with 205 line drawings. London : Baillière, Tindall & Cox. Price 21s.]

" M'Fadyean's Osteology and Arthrology " needs no introduction to the veterinary profession, and the publication of this extensively revised edition after a lapse of almost 30 years, will be welcomed by all interested in comparative anatomy. In this revised edition, the original method of presentation has been retained. The horse is dealt with very fully as the type animal, and the other domestic animals are considered comparatively. The value of this revised and modernised text is greatly enhanced by an extensive range of new illustrations, which are simple and easy to understand and, in addition, include clear diagrams of all articular surfaces. However, many of the illustrations tend to be rather small, especially those of the skull, and so lose much of their clarity. A completely new and useful addition is a note on bone development at the end of each section on the horse. It is felt that the usefulness of the book would have been even greater if the section on arthrology had been extended with special reference to those animals treated comparatively and, in particular, the dog.

In spite of these criticisms, this edition will adequately supply the veterinary student with the essential basic knowledge of bone and joint structure, and all those who obtained value from the previous edition will not be disappointed in the new one.

The book is printed in clear type on a good semi-gloss paper, and is well bound.

CLINICAL COMMUNICATION

THE SURGICAL REPAIR OF THE RUPTURED DIAPHRAGM

JOAN N. MILNES, M.R.C.V.S.
Huddersfield

Case 1. *Subject.*—A seven-year-old female cat.

History.—A gradually increasing difficulty in breathing and complete inappetence.

Clinical Examination—The cat was in an advanced stage of pregnancy with great abdominal enlargement. The temperature was normal. There was acute dyspnoea.

Diagnosis and Proposed Treatment.—As the owner insisted on either destruction or an operation to afford immediate relief, a tentative diagnosis of respiratory embarrassment, due to abdominal pressure was made, and ovaro-hysterectomy advised.

Anaesthesia.—Oxygen ether.

Operation.—A normal ovaro-hysterectomy was performed through a mid-line incision. Due to the large size of the uterine horns, ½ c.c. of posterior pituitrin was injected intramuscularly, to reduce shock, prior to withdrawing the horns from the abdomen. Dyspnoea was still acute. After removal of the uterus, the abdomen appeared somewhat empty. A large diaphragmatic rupture was discovered, with herniation of part of the liver, the stomach, spleen and a considerable quantity of intestine. After extending the original incision anteriorly, these were withdrawn into the abdomen, when there was an increase in the dyspnoea. However, respiration did not cease as was expected. The rupture was sutured using No. 2 silk and a continuous suture for the sake of speed. Two or three times, during the repair of the rupture, air was manually expressed from the chest, and the abdominal incision was nipped together with the fingers quickly, before the next inspiration, and held until the dyspnoea was reduced a little. Just before the suture was completed the air was again expressed from the chest, and the remaining small hole then quickly closed. The abdominal wound was then sutured in the normal manner.

Post-operative Treatment.—An electric fire was placed in front of the cage, and that evening the cat stood and lapped glucose and milk. 300,000 units of penicillin were injected daily for a week, and by the fourth day respiration was normal. From then recovery was uneventful. The cat returned home on the eighth day, and since has led a normal life.

Case 2. *Subject.*—A five-year-old female cat.

History.—After being hit by a car, there had been increasing difficulty in breathing and complete inappetence.

Clinical Examination.—The cat was in an advanced stage of pregnancy. Auscultation showed the absence of respiratory sounds over much of the chest. The temperature was normal.

Diagnosis.—Respiratory embarrassment from abdominal pressure or a diaphragmatic rupture.

Anaesthesia and Operation.—Procedure as in Case 1.

Post-operative Treatment.—As in Case 1, with an equally uneventful recovery.

Case 3. *Subject.*—Male sheepdog, 18 months of age.

History.—Half an hour previous to being seen it had been run over by a lorry and was vomiting blood.

Clinical Examination.—The mucous membranes were white, the heart rapid and weak and the respirations extremely laboured. On auscultation no normal respiratory sounds could be heard in either lung, and X-ray examination showed a rupture of the diaphragm with herniation of viscera into the left side of the chest. There was considerable fluid in both lungs.

Pre-operative Treatment.—For four days the dog was treated for shock and 300,000 units of penicillin given daily. Gradually normal lung sounds became apparent in a few small areas on auscultation.

Anaesthesia.—In view of the success with the two cats two weeks previously, Intraval Sodium was administered to the stage of recumbency only. Anaesthesia was then further induced and maintained by oxygen ether.

Operation.—After the usual preparation of the operation site, and arranging of operation cloths, an incision was made parallel to and ¾ inch away from the last rib on the left side, as for a gastrotomy. This was found to be inadequate as the entire liver was herniated, besides the stomach, spleen and a considerable quantity of intestine. The incision was extended across the mid-line to the right side. The whole hand could then easily enter the chest, and the liver was withdrawn and held outside the abdomen on the right-hand side. This brought the extensive rupture into an easy position for suturing. There was acute dyspnoea with considerable cyanosis, but at no time did respiration entirely cease. Before the rupture was finally closed with No. 3 silk sutures, air was expelled from the chest cavity by manual pressure and the final suture tied off quickly. Two or three times during the operation when cyanosis was acute, the liver was returned to the abdomen, air again expelled by manual pressure, and the abdominal wound held closed by hand until there was considerable relief. The abdominal wound was closed in the usual manner.

Post-operative Treatment.—300,000 units of penicillin were given for a further four days. There was again uneventful recovery, and at the end of the week the dog returned home to a normal life and since has remained fit and well.

Discussion

The three cases have been described with a view to encouraging those veterinary surgeons who do not possess a form of closed circuit anaesthesia, to attempt to save the lives of animals in cases of diaphragmatic rupture, and not treat them as hopeless, as I myself had done previously in bad cases.

REPORT

All-India Society for the Prevention of Cruelty to Animals

No one from the British Isles who has visited India has felt other than unhappy at the gross over-population of domestic animals and the enormous amount of starvation and suffering that goes on unchecked and unheeded. However humane the individual Indian is, he cannot take animal life without at once coming up against the taboos of caste and his religion. This makes the task of the animal welfare worker throughout India well-nigh impossible. Despite these obstacles, many notable and devoted Europeans and a few Indians have endeavoured to do something for the starving, sick and unwanted creatures that infest the towns. The report of the All-India S.P.C.A. is a small beam of light in an otherwise dark environment.

The Society is attempting to co-ordinate the activities of those animal welfare bodies—most of them founded by devoted Europeans—and acts in an advisory capacity generally in the prevention of cruelty to animals. It was founded by Lady Lloyd and it is good to know that her work is being continued. Some 40 local societies are listed as being affiliated to this body, and one can only hope that the work started during the British occupation may gain momentum and strength and become something of real value throughout this great country.

ABSTRACTS

Positive Pressure Anaesthesia and Intrathoracic Surgery in Small Animals*

I.—Positive Pressure Anaesthesia

The author points out that due to the frail mediastinal septum in small animals, artificial respiration is essential for any surgical operation involving pneumothorax. Since the animal's head is not suitable for a well-fitting mask or face piece, endotracheal intubation is necessary and, for this, the human range of Magill-type cuffed tubes are satisfactory. Intubation requires a plane of anaesthesia deep enough to obtain full relaxation of the jaw to permit visual or blind intubation. For this purpose, an intravenous injection of a barbiturate is used, but it is pointed out that in the cat it produces a marked glottic spasm which is best overcome by "Flaxedil" (Gallamine triethiodide). In addition, premedication with a sedative antispasmodic omnopon (pantopon) or pethidine hydrochloride (demerol) with lyosine to suppress mucous secretions is desirable.

In conjunction with Mr. C. R. Wood, M.R.C.V.S., of London, the author has designed an apparatus to convey air intermittently to the lungs for positive pressure ventilation. It is used for small animals in combination with intravenous barbiturate anaesthesia, and enables a variable but controlled tidal volume of air, also oxygen, to be delivered to the lungs.

Alternative methods are discussed briefly. One is insufflation technique, employing a rebreathing bag and semi-closed or closed circuits. These inhalation techniques permit the use of muscular relaxant drugs, but it is pointed out that these drugs permit restoration of consciousness in spite of respiratory and motor paralysis, and care must be taken to ensure that their action is confined to the anaesthetic period.

It is emphasised that controlled respiration is necessary to obtain a stationary field for the surgeon, and the author suggests that the best combination of anaesthesia and positive pressure is obtained with cyclopropane gas. However, two objections should be considered: (i) Its explosive nature which forbids the use of diathermy, and (ii) the susceptibility of dogs to overheating which is enhanced in hot weather by the carbon dioxide absorption techniques.

II.—Intrathoracic Surgery

The author describes three surgical interventions entailing pneumothorax.

(*a*). Transthoracic oesophagotomy. This operation is performed by the author for removing foreign bodies in dogs when critical consideration of all circumstances favours this method rather than extraction with forceps along the oesophagus to the mouth or by gastrotomy. The technique employed is to resect the rib which is superimposed over the foreign body as determined by X-ray. Rib resection is favoured rather than simple intercostal incision, because a better exposure is obtained and occasions less risk of injury to the costal-vertebral articulations. Also, closure is more easily effected by only having to suture adjacent intercostal muscles without the necessity of including adjacent ribs. The incised oesophagus bleeds profusely and care must be taken to arrest this before closure which can be satisfactorily accomplished with continuous or interrupted sutures of No. 3 chromicised 20-day catgut on an atraumatic needle. To expel air from the chest, the lungs are momentarily held fully inflated at about

* **Positive Pressure Anaesthesia and Intrathoracic Surgery in Small Animals.** Knight, Gordon. (1953). Proc. Part I. XVth Int. Vety. Congress, Stockholm. **2.** 953–957.

8 to 12 mm. Hg. to coincide with the tying of the final suture to close the chest.

(*b*). Repair of the ruptured diaphragm. Rupture is most frequently seen in dogs and cats as the result of trauma. In the author's experience, the breakdown has been mostly peripheral, but frequently continuing well into the tendinous portion.

The rupture is invariably approached from the abdomen through a slightly paramedian incision. Extensive adhesions of abdominal viscera to pleura sites have never been encountered. After the abdominal viscera has been returned to the abdominal cavity and all transudate removed by aspiration, the rupture is closed with linen thread interrupted mattress sutures. A technique similar to that employed in transthoracic oesophagotomy is used to exclude air from the chest cavity when suturing of the diaphragm is complete.

(*c*). Exploratory thoracotomy. According to its purpose, the incision may be either a simple intercostal one or rib resection if wide exposure is required. The operation may be necessary for diagnostic purposes such as biopsy, removal of foreign bodies from the pleural cavity, arrest of intrathoracic haemorrhage, removal of blood or transudate.

In conclusion, the author states that the impunity with which the pleural cavity can be exposed to bacterial infection without threat of post-operative sepsis is due largely to the use of the antibiotics.

J. H.

* * * * *

Practical Prosthesis for the Femoral Head*

A high percentage of femoral neck fractures result in complete resorption of the bone and avascular necrosis of the femoral head. Intramedullary pinning has often resulted in failure of the bone to heal.

The authors describe a plastic prosthesis for the femoral head, using materials and methods available to the general practitioner.

The plastics used are methyl methacrylate and vibrin 108, both in liquid form. The vibrin is less critical and requires only the addition of a catalyst before use.

The size of the prosthesis is determined from radiographs of the normal femur. A test tube or gelatine capsule, with an inside diameter equal to or slightly less than that of the femoral head, is used as a mould. A stainless steel pin, equal in length to the distance between the fovea capitis and the great trochanter, is used to strengthen the plastic. One end of the pin fits into the artificial femoral head while the shaft strengthens the stem of the prosthesis.

When hardened the casting is removed from the mould, machined to size and polished. The diameter of the stem should be 1/32 inch larger than the bit used to drill the femur.

The coxofemoral joint is exposed, the capsule severed completely, and the femoral head removed. The proximal end of the femur is exposed, squared and drilled to admit the stem of the prosthesis, which is then driven into position. The plastic head is inserted into the acetabulum and muscle and skin sutured.

Supplementary fixation is unnecessary and the animals bear weight on the operated leg within three days.

L. C. V.

* **A Practical Prosthesis for the Canine and Feline Femoral Head.** Archibald & Ballantyne. (1953). *N. Amer. Vet.* **34**. 496–501.

ROYAL COLLEGE OF VETERINARY SURGEONS

Membership Examinations, December, 1953

List of Successful Candidates

London

Second Examination

Physiology only

Barnett, K. C.
Blackmore, D. K.
Bruford, J. C. (credit)
Cocks, Edwin
Cook, W. E. R.
Davies, Miss G. M.
Edgar, R. K.
Giles, H. K.
Gill, E. A.
Girling, Miss M. J.
Greenwood, Ronald
Hime, J. M.
Hollands, J. G. (credit)
Jones, C. R. V.
Mason, J. A.
Mullen, P. A.
Nelson, E. P.
Newey, R. M.
Noordin, B. K. M.
Oakley, G. A.
Oke, M. A.
Prasad, A. K.
Ranger, M. J.
Rutherford, L. H.
Sutton, J. B.
Swayne, T. C. B.
Teale, M. L.
Volrath, J. P.
Wells, B. T. (credit)

Anatomy only

Bousfield, W. E. D.
Byrne, G. A.
Davison, R. S.
Holland, William
Hughes, J. R.
Jones, W. A.
McKie, Miss E. M. C. E.
Pearce, J. S.
Robbins, G. R.
Stocks, B. M.
Tanner, J. A.
Wilkinson, John

Third Examination

Hygiene only

Baxter, C. J.
Beaumont, S. H.
Bedson, I. F.
Bowles, Jeffrey
Coryn, H. D.
Davies, M. G.
Derbyshire, J. B.
Dier, B. G. F.
Down, B. C.
Evans, E. D.
Fenton, B. K.
Finn, C. A.
Gee, R. E.
Gray, John
Heptinstall, Jack
Hudson, J. R.
Hyde, Miss K. B.
Jones, R. H.
Kerry, J. B.
Lifton, J. M.
Markham, Keith
Melluish, H. K.
Morath, D. E.
Naylor, D. C.
Norman, D. A.
Owen, Geoffrey
Parkinson, K. D.
Peters, J. B.
Pigott, R. C. C.
Prole, J. H. B.
Rawson-Shaw, Miss E. A.
Shrimpton, D. F.
Skipsey, Roger
Stevens, H. R.
Taylor, E. L. G.
Townsend, Miles
Wall, L. J. T.
Ward, Anthony
Watson, Miss D. P.
Whitaker, D. L.
Williamson, D. C.
Wilson, Miss E. D.
Woollett, W. G. W. A.
Wright, M. A.

Pathology only

Alexander, T. J. L.
Arnold, J. H. S.
Booker, J. C.
Cansfield, C. J.
Cawthorne, W. N.
Cullen, C. S.
Davidson, H. M.
Hambrook, C. L. E.
Holmes, J. R.
Keeling, M. D.
Nelson, G. J.
Roberts, D. H.
Russell, G. H.
Smith, Miss L. C.
Stalbow, Michael
Thomas, C. J.
Thursby-Pelham, R. H. C.
Walker, A. J.
Waller, J. F.

Edinburgh

Second Examination

Black, Hugh
Gee, B. D.
Gray, Brian
McHugh, T. M.
Moore, T. E.
Penman, Gordon
Robertson, H. L.
Rodger, J. L.
Rowland, James
Singh, Arimardan
Walker, W. D.

Third Examination

Ashton, P. K.
Hovell, G. J. R.
Howells, G. J.
Humphreys, Medwyn
Jones, J. S. L.
MacKenzie, R. M.
Manktelow, D. B. S.
Parminter, Donald
Taylor, J. C.
Tennant, L. D.

Fourth Examination

Baird, R. G.	McKinley, D. I.
Black, Michael	Mitcheson, J. A. J.
Campbell, H. M.	Peden, A. B.
Chong, S. N.	Rodger, D. S.
Clarke, Miss C. E.	Stokoe, W. M.
Curran, G. D.	Thorne, J. P.
Guilor, Miss Mary	Trainer, P. E.
McAllister, Joseph	Wilson, H. R.
McCullock, A. F.	

Final Examination

Blizzard, S. V. A.	Marwaha, D. R.
McCormick, A. N.	Thomson, K. S.

University of Glasgow Veterinary School

Fourth Examination

Crawford, T. G.	Loudon, A. J. B.
Gracie, D. K.	Martin, Miss Heather
Hutchison, James	

Final Examination

Bickerton, D. T.	Ommer, F. G.
Bradley, J. A. P.	Russell, D. A.
Gilmour, M. J.	Slater, Duthie
Gittleson, M. L.	Stewart, G. H.
Jawara, S. A.	Thom, Alasdair
McCready, P. H.	Towers, Miss I. G.

(To be concluded)

NOTES AND NEWS

BRITISH VETERINARY ASSOCIATION

CHANGE OF ADDRESS

MEMBERS, OTHER READERS AND ADVERTISERS are requested to note that the

ADDRESS OF THE BRITISH VETERINARY ASSOCIATION AND "THE VETERINARY RECORD"

is now

7, MANSFIELD STREET, PORTLAND PLACE, LONDON, W.1.

Telephone number: Museum 6541

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Diary of Events

Jan. 5th.—Extraordinary General Meeting of the South Wales Division, B.V.A., at Carmarthen (The Dolwar Café), 5.30 p.m.

Jan. 6th.—Meeting of the Association of Veterinary Teachers and Research Workers, at the Royal (Dick) School of Veterinary Studies, Edinburgh, 10 a.m.

Jan. 7th.—Meeting of the Central Division, B.V.A., at the Royal Veterinary College, N.W.1, 6 p.m.

Jan. 13th.—Meeting of the R.A.V.C. Division of the B.V.A., at Langley Court, Beckenham, Kent.

Jan. 13th.—Meeting of the Editorial Committee, B.V.A., at 7, Mansfield Street, W.1, 2.30 p.m.

Jan. 14th.—Meeting of the South-Eastern Division, B.V.A., at Maidstone (Royal Star Hotel), 2.30 p.m.

Jan. 14th.—Meeting of the Western Counties Division, B.V.A. at Exeter (The Imperial Hotel), 2 p.m.

Jan. 14th.—First Pharmaceutical Society Meeting to introduce The British Veterinary Codex (*see Notice*).

Jan. 16th.—Meeting of the Biochemical Society, in the Department of Chemical Pathology, Post-graduate Medical School of London, Ducane Road, W.12, 11 a.m.

Jan. 27th.—Meeting of the Supplementary Veterinary Division, B.V.A., at the Wellcome Research Laboratories, Langley Court, Beckenham, Kent, 2 p.m.

Jan. 27th.—Second Pharmaceutical Society Meeting to introduce The British Veterinary Codex (*see Notice*).

Feb. 11th.—Royal (Dick) School of Veterinary Studies Annual Ball, in the Assembly Rooms, George Street, Edinburgh, 8.30 p.m. Proceeds to the Victoria Veterinary Benevolent Fund.

Feb. 26th.—University of Liverpool Veterinary Society Annual Ball, in the Students' Union, Bedford Street, Liverpool.

Change of Date of Next Meetings of Council and Committees, B.V.A.

Owing to the fact that headquarters has moved to 7, Mansfield Street just before Christmas, the meetings fixed for early January (6th, 7th and 8th) have been postponed until Wednesday, Thursday and Friday, January 20th, 21st and 22nd, 1954.

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R.C.V.S. Council Election, 1954

Nomination forms, to be obtained from the Registrar, 9, Red Lion Square, W.C.1, should be returned not later than February 1st, 1954.

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PERSONAL

Birth.—Norman.—On December 22nd, 1953, to Anne, wife of Thomas H. Norman, B.Sc., M.R.C.V.S., of Kington, Herefordshire, a daughter—Susan Elizabeth.

. . . . .

Appointment.—Mr. Richard T. W. Fiennes, B.A., M.R.C.V.S., leaves this country on January 14th to take up an appointment as Senior Lecturer in Animal Diseases with the Department of Agriculture of the University of the Gold Coast.

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R.C.V.S. OBITUARY

Minett, Francis Colin, C.I.E., M.B.E., D.Sc. (Lond.), M.R.C.V.S., Captain R.A.V.C. (retd.), after a long illness, at Windybank, Gorse Way, Hartley, near Dartford, Kent. Director of the Animal Health Trust's Farm Livestock Research Station, Houghton Grange, Hunts; formerly Director of Veterinary Research Institutes in London and Mukteswar, India; also Animal Husbandry Commissioner to the Government of Pakistan. Correspondant étranger de l'Académie royale de Médècine de Belgique; Correspondant étranger de l'Académie de France. Graduated London, September 16th, 1911. Died December 26th, 1953; aged 63 years.

. . . . .

Death of Dr. F. C. Minett, C.I.E., M.B.E., D.Sc., M.R.C.V.S.

We record above with the deepest regret the death of Dr. Francis Colin Minett, which has occurred but a relatively short while after this distinguished worker in the field of veterinary research, whose experience was great and whose renown was wide, had settled down to the scientific direction of the Animal Health Trust's Farm Livestock Research Station in Huntingdonshire. Supreme indeed has been the fortitude with which Dr. Minett has faced the inevitable outcome of a condition manifested in the late summer, and the sense of loss will be the more widespread because of the immense range of his activities, to be revealed in the memoir which we hope shortly to publish. In summary, we may remind our readers that, having served with the R.A.V.C. and retiring as Captain with the M.B.E. in 1916, Colin Minett worked as a Research Officer at Weybridge before becoming Director of the Research Institute in Animal Pathology at Camden Town. In 1932 he was appointed Professor of Pathology in the College, a post which he held until 1939, when he went to India as Director of the Imperial Veterinary Research Institute. In 1947 his life work on the pathology and bacteriology of disease conditions of farm livestock, which he had recorded in upwards of 50 original articles, found natural fruition in his appointment as Animal Husbandry Commissioner to the Government of Pakistan. From this post he retired in 1949, taking up his appointment with the Animal Health Trust in the following year.

Personal Tributes

Dr. W. R. Wooldridge, Scientific Director of the Animal Health Trust, writes:—

Members of the veterinary profession and many in allied sciences who knew him will mourn Dr. Minett's comparatively early death, for the loss of one who has maintained such high standards of scientific endeavour can be ill afforded. His work in the realms of bacteriology and pathology will assuredly be discussed elsewhere in your columns. Had he belonged to another profession, whose connections with the Royal Society more easily fitted into the ancient classification by which scientists are elected into its fellowship, Minett would almost certainly have received this honour: assuredly his work appeared to many to deserve it.

I would rather speak of Minett as one whom I came to know intimately during the last few years. Many years ago, when President of the Veterinary Research Club, I had the privilege of nominating Minett for its membership—thereby rectifying an earlier omission. I then had many long discussions with him concerning professional progress, and whilst he felt deeply and sometimes personally the unfortunate groupings within the academic side of the profession, he agreed to assist the Club to overcome them. From then onwards until he went to India, Minett became one of its most staunch supporters and a very severe but just critic of the many scientific contributions read to the Club.

Later, when Minett returned from India, I had the further privilege of becoming one of his colleagues when he became Director of the Farm Livestock Research Station of the Animal Health Trust. Unfortunately events prevented him from fulfilling our hopes in this direction. Firstly, when we were asked on behalf of the Foreign Office to allow Minett to go to Turkey to advise the Government of that country on its veterinary services, our plans in the Trust were held up for more than a year. Then on his return it became evident within a few months that Minett was far from well. At long last he was persuaded to take proper advice upon the cause of his illness. He underwent a severe operation which at first seemed successful, but all too soon his illness redeveloped. Even so, during this difficult and chequered period my colleagues and I got to know Minett well and at every stage our admiration for him increased. His critical scientific ability, his determination, his fortitude, and his courage will ever inspire us.

Beneath an apparently austere exterior Minett was a most warm-hearted individual. To many he may have seemed to be comparatively devoid of humour. On the contrary he was full of fun and bubbling over with warm-hearted friendliness to those who were privileged to know him well. In some ways his life had not been as happy as many would have wished, but Minett overcame his own troubles and devoted himself with patience and enthusiasm to his work. Minett was convinced that the best way in which he could advance the cause of the profession was to make and encourage researches of high scientific standard.

Personally I shall always cherish the friendship which developed between us and shall hope that his high scientific ideals have been absorbed both by me and by many other members of the profession.

* * * * *

In the passing of Minett we lose another member of what one may describe as the "first generation" of British veterinary research workers. Minett was pre-eminently a research worker in the best sense of the word; he was only interested in research work for itself and this single-minded attachment to the actual doing of the work himself will always be remembered by the writer as a source of inspiration. Administrative duties and directorial responsibilities were never allowed to interfere with his personal research work and this in itself was an example to young workers in his institute. Others will write of his very considerable contributions to veterinary knowledge, but as a worker on mastitis I shall always remember that it was the work of Minett and his two junior colleagues, Stableforth and Edwards, which cleared the bacteriological jungle and allowed mastitis to be studied in a logical and proper manner. Minett was a man with whom one had to have frequent contacts to appreciate his true worth; on superficial acquaintance his manner was brusque to say the least, but underneath was a scientific integrity and a persistence and courage of character which will always be remembered by the writer. The last occasion on which I saw Minett was about a month ago when he was extremely ill, sinking rapidly, and fully aware of the implications of his condition, and yet he had only finished writing on the previous day a scientific article and a contribution to a book. I shall always be grateful to him as my first chief and as the man who gave me an example of the importance of research work and the need continuously to engage in it oneself, and also for the example of his great courage in the last few months of life. D. L. H.

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Between the years 1927 and 1939, when Dr. F. C. Minett was Director of the Research Institute, Royal Veterinary College, London, it was my privilege to act as his personal technician and since to have had his friendship and counsel. During these times I came to know him well, probably better than most. His mind and thoughts were devoted to research work, particularly then with the subjects of mastitis and Johne's disease, and his enthusiasm encouraged those that worked with him to greater efforts.

He never spared his energies, his days were long and arduous and at times when he was baffled or encouraged he would continue to work far into the night. It was quite common to find him working either in the laboratory or library until the early hours of the morning.

Dr. Minett possessed the art of planning his work for days and weeks in advance in order that no time should be wasted or the results of an experiment delayed. Administrative duties would not cause him to deviate from this plan and in order to prevent this happening he would abandon his own spacious and well-equipped laboratory to seek quietness for his research in a smaller room, where the door would be locked against all.

The knowledge and technical experience that one gained from association with him in his labours have proved of immense value and one feels a debt of gratitude for his help and guidance.

The power he possessed to extract the utmost from his colleagues was not only apparent in his work, for he was a staunch supporter of sport and a very active member of the social life amongst the staff at college. He was extremely keen on cricket and played on numerous occasions, when he would insist in taking the position of wicket-keeper. This involved him in many injuries, but he was never more proud than when he could display evidence of his attempts to stop the fast bowlers. He drove his fellow players to give of their best and continually called for more speed in the field.

Dr. Minett was always mindful of junior members of his staff and would often discuss with me the possibilities of giving help. I can recall his generosity on many occasions, and I know that those who were with him in London will long remember him and respect his memory.

He bore his illness with amazing fortitude and until the last made no mention of his unfortunate lot but only had thought for others. He sought solace in writing what he termed "the finishing up of tail-ends of work which time had not allowed him to do before."

We mourn his going but shall treasure his memory. He has left as his monument the work so ably done during his lifetime.

A. E. Mundy,
Boots Pure Drug Co. Ltd.,
Veterinary Science Division,
Thurgarton, Notts.

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ART AND MEDICINE

Paris Invites British Artists

The 26th Exhibition of Paintings by Doctors, Pharmacists, Dentists and Veterinary Surgeons (Le 26e Salon des Médecins, Pharmaciens, Dentistes et Vétérinaires) will be held in Paris from March 7th to 21st, 1954, at the Musée d'Art Moderne (France's National Art Museum and Gallery), 11, Avenue du Président Wilson.

As in past years, it will group sections for paintings, sculptures, photography and applied decorative art. After the great success the salon has enjoyed, it is desired to bring the 1954 exhibition up to an even higher standard. Colleagues are invited to send their works of art, vacation and pleasure to this Paris exhibition. The Minister of Public Health will again reward the laureates who will receive medals.

Although the salon is a French one, in view of the Franco-British Cultural Relations Campaign contributions from British artists are cordially invited from the British medical and veterinary professions.

Works, etc., should not be sent direct to the salon, but should be addressed to the forwarding agents, Messrs. Davies Turner, Ltd., 4, Lower Belgrave Street, S.W.1, to reach London not later than February 10th, 1954. Each object must bear a label giving full particulars: name, address, qualifications (Dr., Dent., Pharm., Vet. Sur.) and title of the exhibit. Those wishing to participate should inform the Secretary, Dr. Malet, 67, Ave. Pierre-Larousse, Malakoff (Seine), France (Tel. ALEsia 0709), not later than February 10th, 1954, with full particulars as above, in order that these may appear in the catalogue.

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BRITISH STANDARD FOR RECOMMENDED COMMON NAMES FOR PEST CONTROL PRODUCTS

The British Standards Institute has recently issued Part 2 of the British Standards Recommended Common Names for Pest Control Products. This list results from the work of a committee representing various industrial and other associations and societies. The British Veterinary Association is represented on this committee. The first list of recommended common names for pest control products was published in 1952 and the present pamphlet lays down standard names for a further series of insecticides, fungicides and rodenticides which have been considered since the publication of the first part.

The numbering of the common names is a continuation of that adopted in Part 1.

Copies of this British Standard (B.S. 1831, Part 2: 1953) may be obtained from British Standards Institution, Sales Branch, 2, Park Street, London, W.1. Price 2s. net.

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CONFERMENT OF DEGREES ON MEMBERS, R.C.V.S., BY THE UNIVERSITY OF EDINBURGH

The following degrees have been conferred by the University of Edinburgh upon members of the Royal College of Veterinary Surgeons:—

At a Graduation Ceremonial held on October 24th, 1953:
Bachelor of Science (Veterinary Science): T. H. Norman, M.R.C.V.S.

At a Graduation Ceremonial held on December 18th, 1953:
Doctor of Science (in the Department of Veterinary Science): Archibald McDiarmid, PH.D., M.R.C.V.S. Thesis: "Immunological Studies on Brucellosis with Special Reference to the Use of S.19 Vaccine in Cattle."

Doctor of Philosophy (in the Faculty of Medicine): W. C. D. Hare, B.SC., M.R.C.V.S. Thesis: "A Study of the Distribution of the Bronchial Tree, the Pulmonary Artery and the Pulmonary Vein in the Lungs of the Sheep."

Bachelor of Science (Veterinary Science): S. V. A. Blizzard, M.R.C.V.S.

All the above-mentioned graduates qualified at the Royal (Dick) School of Veterinary Studies; Dr. Hare is a Lecturer in Veterinary Anatomy in the school. Dr. McDiarmid is on the staff of the A.R.C. Field Station at Compton.

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UNIVERSITY OF LIVERPOOL DEGREES IN VETERINARY SCIENCE

Following is the December, 1953, Examination List for the University of Liverpool Faculty of Veterinary Science:—

DEGREE OF B.V.Sc.

SECOND EXAMINATION: PART III

Crabb, P. J.
Lancaster, R.
Murgatroyd, A. E.
Ward, R. K.
Wortley, G. H.

THIRD EXAMINATION: PART II (*Old Regulations*)

Arnall, L.
Brown, J. K.
Farrer, J. S.
Haworth, E.
Jaggar, J. H.
Kneen, J. E.
Rees, J. B.
Rothwell, J.
Sanders, G.
Taylor, Patricia

DEGREE OF PH.D.

Platt, H.

DEGREE OF M.V.Sc.

Field, A. C.

TRANSFORMATION IN THE MEDICINAL TREATMENT OF DISEASE

Sir Henry Dale, O.M., F.R.S., delivering the first of the 1953 Harben lectures at the Royal Institute of Public Health and Hygiene, London, said that it was planned to devote the lectures to a review of the remarkable changes which, during the past half-century, and largely in the recent quarter, had so transformed the whole aspect of the medicinal treatment of disease.

The beginning of the change could be associated with the recognition of the removal of the causes of disease as a proper and practicable aim of medicinal treatment, whereas it had earlier not been expected to do more than ameliorate symptoms. The origin of this new aim could be traced to the earliest years of the 1890s, when Emil von Behring discovered the diphtheria and tetanus antitoxins, Paul Ehrlich made his first, though not practically successful, trial of chemotherapy, and George Murray successfully treated a disease of deficiency, myxoedema, by administering thyroid gland, containing the deficient hormone. Recent developments seemed to indicate the extension of this more scientific approach to the newer types of remedies dealing with symptoms.

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ADDRESSES OF DISEASE-INFECTED PREMISES

The list given below indicates, first, the county in which are situated the premises on which disease has been confirmed, followed by the postal address and date of outbreak.

FOWL PEST:

Essex.—Westbury Poultry Farm, Downham, Billericay (Dec. 19).
Glam.—Railway Sidings, Treforest, Pontypridd (Dec. 19).
Norfolk.—Holly Farm, Bressingham, Diss (Dec. 20).
Yorks.—Grange Farm, Headingley, Leeds (Dec. 20).

SWINE FEVER:

Cambs.—15, Fulbourn Road, Cherry Hinton (Dec. 19).
Essex.—Eight Acres Farm, Sewardstone Road, Chingford (Dec. 19).
Flints.—Wolvesacre Hall, Iscayd, Flint (Dec. 19); Tynewydd, Cyman, Wrexham, Denbighs (Dec. 21).
Glam.—15, Blaencaeran Road, Caeran, Maesteg (Dec. 19).
Glos.—Woodleaze Farm, Horsley, Stroud (Dec. 22).
Kent.—219, High Street, Rainham, Gillingham (Dec. 21).
Salop.—Horton House Farm, Horton, Wem, Shrewsbury (Dec. 22).
Suffolk.—Toad Row Farm, Henstead, near Beccles (Dec. 22).
Surrey.—Peartree Cottage, Folly Hill, Farnham (Dec. 19); Island Farm, High Street, West Molesey (Dec. 21).
Staffs.—5, Golds Hill, West Bromwich (Dec. 21).
Yorks.—14, Camm Lane, Mirfield, York (Dec. 21).

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AIR ACTION AGAINST LOCUST SWARMS

Reuter's Correspondent at Nairobi reports the departure, on December 22nd, of 10 light aircraft from Nairobi for the northern frontier province to provide a new weapon against an expected invasion of East Africa by millions of locusts, which at present are breeding over an area of 150,000 square miles—most of it dense bush—in the Somalia peninsula. A total of 35,000 gallons of concentrated insecticide has been placed in strategic dumps on the northern frontier for use from the air. Though it has been tried experimentally, this will be the first time that air spraying has been used operationally against locusts.

CORRESPONDENCE

The views expressed in letters addressed to the Editor represent the personal opinions of the writer only and their publication does not imply endorsement by the B.V.A.

SMITH'S "VETERINARY PHYSIOLOGY"

Sir,—Will you kindly allow me a little of your space to thank the numerous members of your Association who offered me copies of Smith's "Veterinary Physiology." One even got on the telephone to me before 9.30 a.m. of the day my letter was published to say that he had a copy of this book to which I was welcome.

I am most grateful to you and to all those who wrote to me, to whom I am replying individually.—Yours faithfully, R. F. WEST, Director, Baillière, Tindall & Cox, Ltd., 7 & 8, Henrietta Street, Covent Garden, London, W.C.2. *December 23rd, 1953.*